“STUDY OF FETOMATERNAL OUTCOME IN GESTATIONAL DIABETES MELLITUS’’

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MAY - 2018

CERTIFICATE

This is to certify that the dissertation titled **" STUDY OF FETOMATERNAL OUTCOME IN GESTATIONAL DIABETES MELLITUS"** is a bonafide work done by **DR.V.NITHYA** in **CHENGALPATTU MEDICAL COLLEGE,** during the academic year 2015-2018 submitted to the **TAMILNADU Dr.M.G.R.** Medical University in partial fulfillment of University regulation for M.S. Branch - II Obstetrics and Gynaecology degree examination of The TamilnaduDr.M.G.R Medical University.

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DECLARATION BY THE CANDIDATE

I,DR.V.NITHYA, solemnly declare that the dissertation titled **"STUDY OF FETOMATERNAL OUTCOME IN GESTATIONAL DIABETES"** has been prepared by me. I also declare that this bonafide work or a part of his work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad. This is submitted to The Tamilnadu Dr.M.G.R.Medical University, Guindy, Chennai in partial fulfillment of the rules and regulation for the award of M.S.degree Branch- II Obstetrics and Gynaecology.

Place:Chengalpattu **Signature of the candidate**

Date: **[DR.V.NITHYA]**

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I humbly submit this work to the Lord Almighty who has given me the health and ability to complete this thesis.

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INSTITUTIONAL ETHICAL COMMITTEE

CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU

Title of Work	:	**Study of fetomaternal outcome in gestational diabetes mellitus**
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The request for an approval From the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 25.10.2016 at the Medical Education Unit, Government Chengalpattu Medical College, Chengalpattu at 12.00 PM.

The Members of the committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

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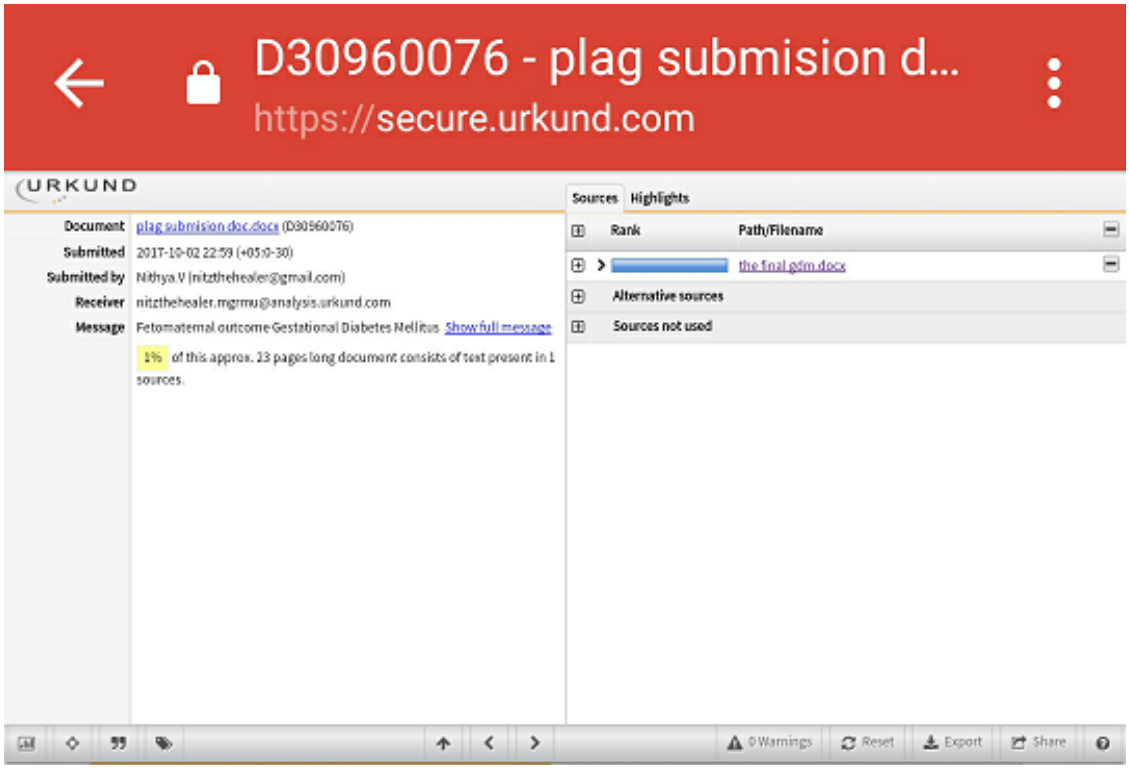

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CONTENTS

INTRODUCTION

Gestational diabetes mellitus means hyperglycemia in pregnancy.By definition it is “carbohydrate intolerance with onset or first recognition during pregnancy”[1]. In recent times there is increasing prevalence of pregnancies complicated by gestational diabetes mellitus. The worldwide prevalence ranges between 11-14%. The prevalence is slightly higher in the Indian population (16.5%) [2], as we Indians are inherently more vulnerable to get affected owing to our hereditary and genetic make up and ethnicity.

The condition occurs exclusively in the antenatal period where there is certain physiological maladaptation in the regulation of carbohydrate metabolism in pregnancy that turns out to be pathological, contributing to the onset and progression of the condition. It can cause a wide range of complications as well as long term implications in both the mother and fetus. The severity of the condition should not be overlooked. The International Diabetic Federation found that one out of seven births in India is affected by GDM. A large proportion of women also progress to become overt diabetics in the future hampering their quality of life by causing morbidity in various forms.

This study was undertaken to evaluate the maternal and fetal complications and postpartum glucose intolerance in patients with Gestational Diabetes Mellitus Chengalpattu Medical College Hospital, Chengalpattu in a period of 1year from October 2016 to September 2017.

AIMS AND OBJECTIVE

- To study the prevalence of antepartum ,intrapartum and postpartum complications in patients with Gestational diabetes mellitus
- To study the outcome of pregnancy in patients with Gestational diabetes mellitus
- To study the incidence of patients with glucose intolerance/overt diabetes mellitus during the postpartum follow up of patients with Gestational diabetes mellitus

REVIEW OF LITERATURE

The first prospective study on metabolism of carbohydrates in pregnancy was established in Boston in 1954,by using 50 gram 1 hour screening test [3]. At that time,the US emphasis was on establishing criteria for the 100g Oral glucose tolerance test in pregnancy as an index of subsequent risk of the mother developing established Diabetes, and the well known O'Sullivan criteria were derived on this basis[2].O'Sullivan christened the name "Gestational Diabetes Mellitus". Formerly it was called metagestational diabetes mellitus[4]. Jorgen Pederson used the term "Gestational Diabetes"in his monograph[5] in 1967,but branded the mother to have gestational diabetes mellitus only after delivery.He found out that abnormal tolerance to carbohydrates especially glucose returned to normal in the postpartum period. The enthusiasm of the team led by Norbert Freinkel and subsequently by Boyd Metzger has ensured that the concept of Gestational diabetes is firmly imprinted on the obstetric mind,as well as having established a major place as an epidemiological tool to study not only the immediate outcome of pregnancy but also the long term effects on both mother and baby of the relatively short phase of hyperglycemia during the later part of pregnancy.

Peter Damm[6] studied the prognosis of women with GDM in previous pregnancy with respect to subsequent development of diabetes and also of predictive factors for developmentof overt diabetes in these women. Insulin

sensitivity in glucose tolerant non obese women with previous GDM were compared with controls. It was found that even these women are characterized by metabolic profile of type 2 Diabetes. Therefore all women with previous history of GDM should have regular assessment of their glucose tolerance in their years after pregnancy.

FREINKELS CONCEPT OF METABOLIC REGULATION IN PREGNANCY

Freinkel studied that there was a state of accelerated starvation in pregnancy[7] and greater than normal levels of ketonemia and ketonuria could hamper the normal fetal development. Therefore strict dietary manipulations like marked restriction of calorie intake should be avoided as it can enhance ketogenesis.

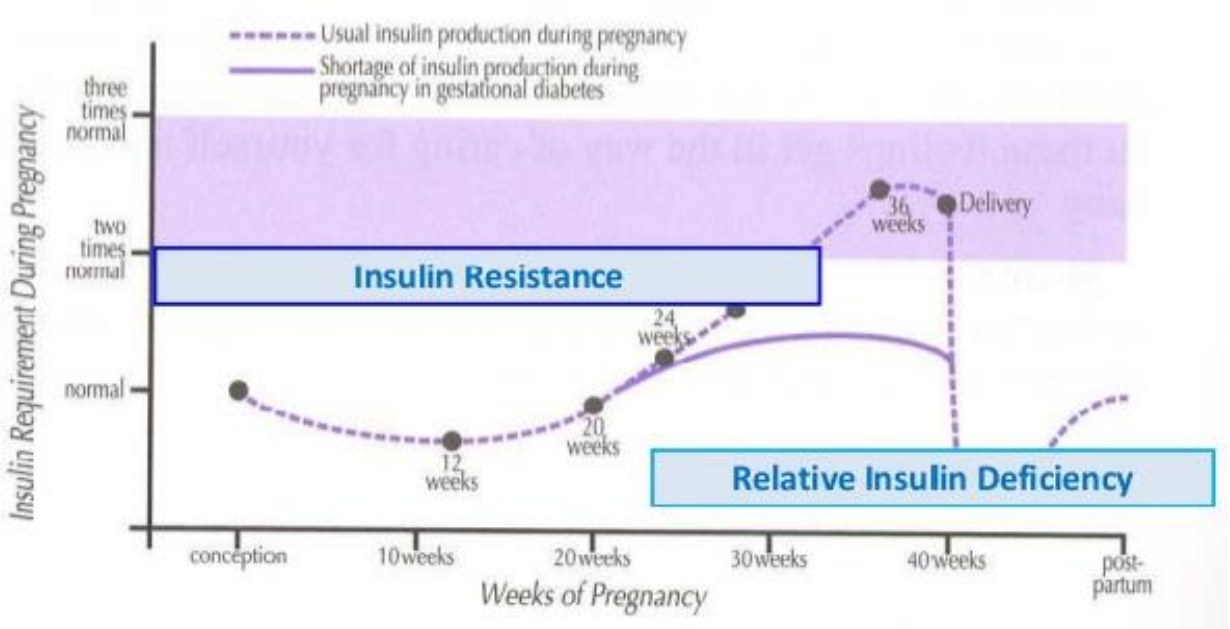
PATHOGENESIS OF GESTATIONAL DIABETES MELLITUS

In majority of the women,insulin resistance increases as pregnancy advances creating demand for more insulin. In the majority,the insulin requirements are readily met so balance between insulin resistance and supply is balanced. When resistance takes the upper hand due to impaired secretion of insulin, hyperglycemia sets in. As in non-insulin dependent diabetes mellitus ,GDM is associated with both impaired secretion and resistance [8]. The two

disorders share the same risk factors and have the same genetic susceptibility. They can be assumed to be etiologically indistinct with one preceding the other.

INSULIN SENSITIVITY AND RESISTANCE IN PREGNANCY

The development of resistance to the glucose lowering effects of insulin is normal during pregnancy. Burt [9] demonstrated that pregnant women experience fewer hypoglycemic events to insulin infusion when compared to non-gravid women. Buchanan et al[10] and Cousins [11] et al demonstrated significant decrease in insulin sensitivity during second trimester of normal pregnancy with a return to normal values shortly after delivery. Ryan et al [12] was the first to report quantitative differences in insulin sensitivity between normal and diabetic pregnancies. .Kalhan et al and Cowett et al [13] noted that hepatic glucose production was increased in patients with GDM than in control groups.



OESTROGEN AND PROGESTRONE

In early pregnancy maternal estrogen and progesterone increase causes beta cell hyperplasia and increase the secretion of insulin[14.] There is increase in peripheral glucose utilizationand glycogen storage with reduction in hepatic glucose production cause fasting hypoglycemia. As pregnancy advances these hormones lead to increased peripheral resistance in the tissues. The anti-insulin activity of progesterone peaks by 32 weeks of gestation [15]

CORTISOL

Cortisol levels increase as pregnancy advances and by the end of pregnancy concentrations are higher by three fold when compared to non-pregnant women[16]. Excess glucocorticoid is characterized by decreased total tyrosine phosphorylation of insulin receptors and is related to post receptor mechanism causing increased insulin resistance[17]

HUMAN PLACENTAL LACTOGEN

The hormone levels increase at the beginning of second trimester. It brings about a decrease in phosphorylation of Insulin receptor substrate and is responsible for profound insulin resistance [18]

LEPTIN

Fasting insulin and leptin concentrations correlate with the body fat making leptin a good marker for obesity and insulin resistance. Leptin can be

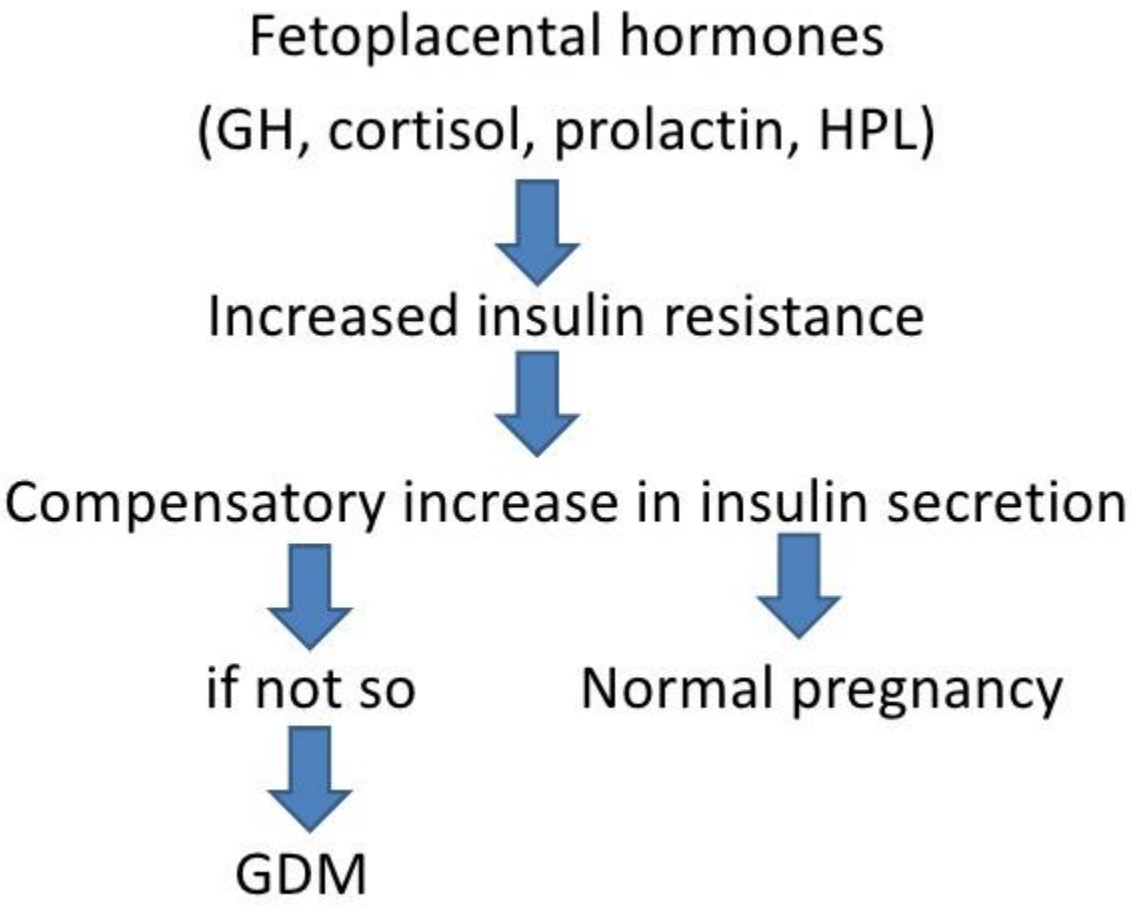
responsible for both central and peripheral insulin resistance. Its levels are significantly higher in pregnancy markedly during second and third trimesters. Alterations in placental leptin levels can contribute to fetal growth independently of maternal glucose levels. Leptin levels are elevated in GDM and can predict the severity of diabetes. Wiznitzer et al [19]reported the umblical cord leptin level was an independent risk factor for fetal macrosomia in non diabetic pregnant women.

TUMOUR NECROSIS FACTOR ALPHA

It has been found to contribute to the pathogenesis of GDM as it is implicated in the regulation of glucose and lipid metabolism. Catalano et al [20]reported that changes in insulin sensitivity from early to late pregnancy paralleled the gradual increase in TNF Alpha levels.

ADRENOMEDULLIN

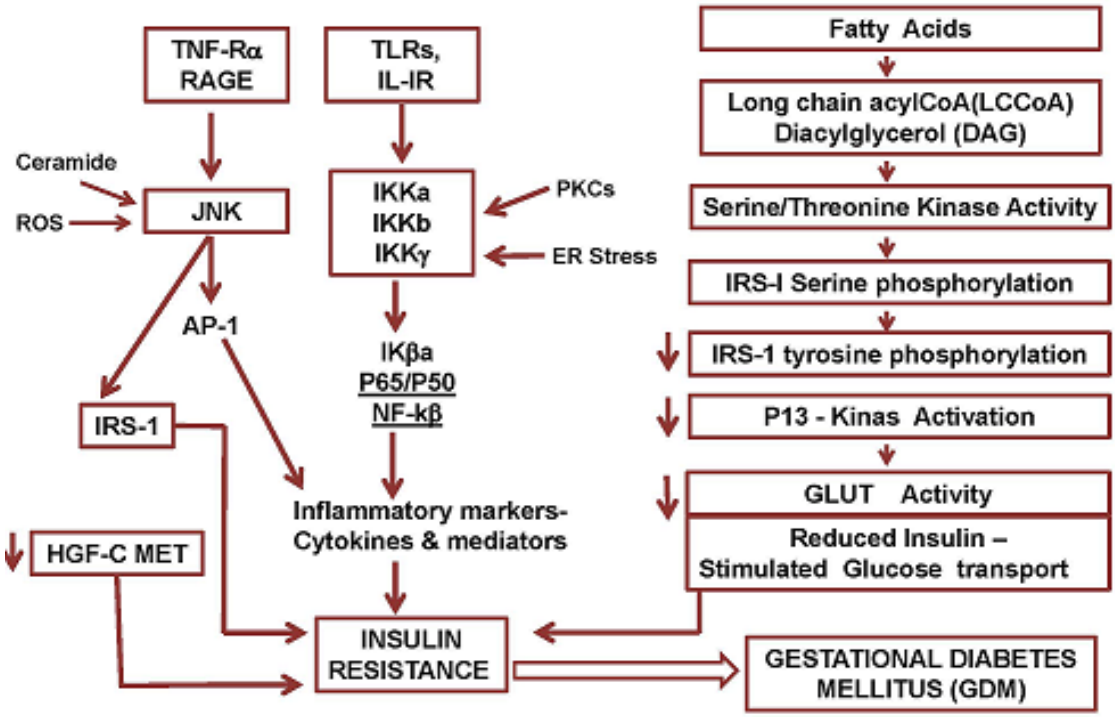
It is a peptide hormone involved in insulin regulatory system. Its amniotic fluid concentration was elevated in pregnant diabetic women when compared to the non diabetic women.



DECREASED BETA CELL RESPONSE

During normal pregnancy, oral and intravenous glucose tolerance decreases only slightly despite the reduction in insulin sensitivity. This is due to gradual increase in secretion of insulin by the beta cells. This happens because of a combination of beta cell hyperplasia and hypertrophy [21]. In GDM,the early insulin response to OGTT is reduced compared to non-diabetic women suggesting a defect in beta cell response. First phase beta cell responses to glucose infusion in GDM are also reduced. Women with normal beta cell function are at lower risk for developing diabetes [22]

To sum up,the dominant pathogenic factor in GDM is a combination of insulin resistance and decreased insulin secretion. Pregnancy is associated with profound hormonal changes that have a direct effect on carbohydrate tolerance. In most subjects, pancreatic insulin secretion increases to meet this need,but in those with underlying beta cell dysfunction,hyperglycemia ensues. Results also suggest that increase in insulin receptor serine /threonine phosphorylation and PC-1(Plasma cell membrane glycoprotein) could underlie the insulin resistance of pregnancy and contribute to its pathogenesis.



The metabolic and endocrine changes in the second half of gestation inducing physiological pregnancy related insulin resistance worsen the underlying metabolic disturbances leading to full blown GDM. Impaired first phase

secretion of insulin,increased second phase insulin release,increased hepatic glucose output relative to prevailing hyperinsulinemia,reduced insulinogenic indices,reduced glucose absorption from the gut,changes in insulin kinetics play an important role in the pathogenesis of GDM.

Other metabolic changes include increased release of amylin and pro insulin that could be a cause or consequence of beta cell dysfunction[23.] Increased intrahepatic and intramyocellular mass and impaired flux through myocellular ATP synthase has also been found in patient with GDM [24]. Impaired lipid metabolism with increased FFA concentrations is also noted. Impaired insulin receptor phosphorylation and insulin receptor tyrosine kinase activity further contribute to metabolic disturbances in GDM[25]. Increased osteoclastin levels in GDM could be regarded as compensatory mechanism to cope up with increased demand on insulin secretion,decreased osteopontin levels relate to decrease levels in GDM.

PLACENTA IN DIABETIC PREGNANCY

Improvements in glycemic controls in the recent yearshave led to less pronounced placental weight differences between normal and diabetic pregnancies. The occurrence of placentomegaly as a result of increased parenchymal tissue cellularity reflected by higher DNA contents confirms a close relationship of placental weight to that of the offspring[26] . There is enlargement of villous surface by 30-50% and increase in the total capillary

surface area. There is increased trophoblastic membrane thickening and hyperplasia of the placental endothelial cells. The placental barrier function is altered [27]

Uteroplacental blood flow may be compromised due to

a)inadequate opening of spiral arterioles by shallow invasion of trophoblast

b)Narrowing of placental bed lumen because of fibrinoid necrosis and foam cell deposition [28]

c) Enlargement of placental villi resulting in decreased intervillous space volume

d)Imbalance in prostacyclin /thromboxane production favoring thromboxane

e) Hyperglycemia induced reduction in trophoblastic estradiol production which has apotent effect on uterine vasculature,hence decreased intervillous perfusion [29]

AMNIOTIC FLUID IN GDM

Hydramnios in GDM has been reported to be as high as 26% which is 40 times that found in non-diabetic controls[30]. This can be extrapolated from osmotic diuresis of hyperglycemic diabetic adult to fetus the hyper glycemic diabetic mother. There was a significant association between amniotic fluid and maternal glucose concentration[31]. In one study, a cohort of 41 insulin treated

women hospitalized for glycemic control for one month underwent amniocentesis prior to elective delivery at 38 weeks. Compared to non-diabetic cohort these demonstrated higher amniotic fluid glucose levels and increased amniotic fluid index. These observations suggest the effect of maternal diabetes upon fetal urine production may require a chronic stimulus associated with maternal hyperglycemia.

Reiher et al[32] demonstrated positive immunohistologic staining for insulin in embryonic fetal pancreas at 9 weeks in pregnancy complicated by diabetes. The fetuses of non-diabetics do not appear to release insulin in response to acutely induced fetal hyperglycemia. Subsequent studies have suggested that prior to mid pregnancy the fetus may produce insulin in women who are at a high risk of subclinical glucose intolerance. Weiss found that third trimester samples of amniotic fluid insulin correlated with birth weights independent of the degree maternal glucose intolerance.

Strangenberg et al[33] found an association of amniotic C-Peptide in third trimester specimens with birth weight suggesting fetal insulin production / renal clearance as manifest in amniotic fluid concentrations are biologically more proximate to pathogenic processes producing diabetic fetopathy. C-Peptide is released in equimolar amounts with insulin. Unlike insulin that is cleared by the liver and kidney, C-Peptide is cleared primarly by the kidney. Consequently amniotic fluid C-Peptide correlate better with fetal insulin release in the third

trimester in GDM pregnancies. Arginine stimulated amniotic fluid C-Peptide and insulin have been associated with fetal macrosomia among insulin treated diabetic pregnant women[34]

To sum up GDM is associated with increase amniotic fluid volume. Amnoitic fluid insulin and C-Peptide are primarily excreted from the fetus there by reflecting fetal pancreatic development in response to intrauterine environment. Late pregnancy amniotic fluid insulin concentrations correlate with the diabetic status. Late pregnancy amniotic fluid insulin and C-peptide levels are associated with clinical signs of diabetic fetopathy in the neonatal period.

DIABETES FOLLOWING GESTATIONAL DIABETES MELLITUS

Although glucose tolerance returns to normal in majority of GDM shortly after delivery,there is substantial evidence that they have an increased risk of developing overt diabetes mellitus in future. O Sullivan found a 36% incidence of diabetes in women with previous history of GDM 22-28 years after pregnancy [35,36,37].

For the diagnosis of GDM, different diagnostic criteria have been employed. In majority of the studies GDM was diagnosed during pregnancy. In the study from Aberdeen GDM was assumed to have occured based on the findings of an abnormal intravenous glucose tolerance test during post partum

period. This tends to select GDM women with relatively more profound metabolic aberrartions since glucose tolerance normally improves shortly after pregnancy. In contest to most of the studies, O Sullivan only included GDM subjects with a normal glucose tolerance after pregnancy in accordance with the GDM definition used at that time .

At follow up, majority of the studies applied 75g oral glucose tolerance test evaluated by the WHO. Not all women with GDM during a specific period are available for participation in a follow up examination several years later. It is important that the women who are investigated at follow up constitute a representative and a large subset of initial GDM population to ensure that the study material is no skewed in anyway that can affect the results. Only few studies give information regarding the participation rate and representativity of the study participants . It has been documented that GDM women with signifcantly increased fasting plasma glucose at diagnosis and those who are treated with insulin have increased risk of developing overt diabetes later in life than those treated with diet alone[38,39] . When comparing follow up studies it is important to know what the treatment or the metabolic status was during the index pregnancy. The current GDM definition allows women with undiagnosed type 2 diabetes antedating pregnancy to be categorised as having GDM. Thus in population with high incidence of these women, a relatively high rate of abnormal glucose tolerance in the post partum period may be found.

The incidence of overt diabetes in the general population is increasing with age. Hence the time span between the index of pregnancy and follow up examination should be considered.

In the Copenhagen study [40], all women with GDM treated with meal plan or insulin were followed up postpartum with a six year median observation time since the index of pregnancy and 34.4% of them had abnormal glucose tolerance in the 2 month postpartum period out of which 13.7% developed diabetes. The incidence of abnormal glucose tolerance seems to increase with increasing follow up time since pregnancy.

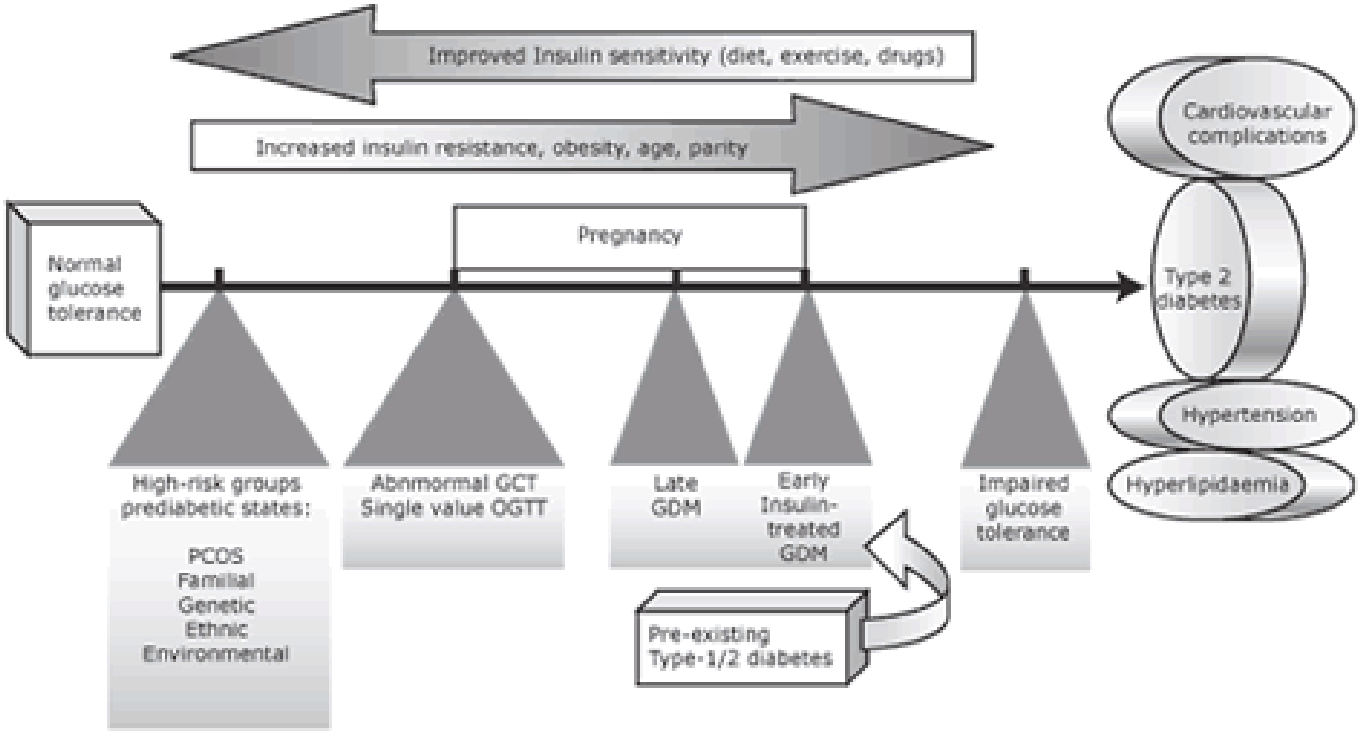
Predictive factors for the development of overt diabetes in women with GDM :

Having confirmed that women with GDM are at risk for subsequent development of overt diabetes it is important to predict who among these women are at highest risk. The following are the risk factors for the development of diabetes or abnormal glucose tolerance during and after the postpartum period in women with GDM.

- Increasing maternal age
- Maternal overweight
- High fasting glucose at GDM diagnosis
- Low fasting insulin at diagnosis
- Low C- peptide during OGTT at diagnosis

- Insulin treatment during pregnancy
- Poor Beta cell compensation for insulin resistance during pregnancy
- Low gestation age at diagnosis of GDM

  The majority of GDM women who have an abnormal glucose tolerance during the postpartum period (although not overtly diabetic) will normalise their glucose tolerance within one year [41]. These women have a more disturbed glucose metabolism compared with women with normal glucose tolerance in the postpartum period and are expected to have increased risk of development of diabetes in later life.



RISK FACTORS OF GDM :

Various risk factors have been identified for gdm and they have stratified to low risk, intermediate risk and high risk (4^{th} international workshop conference on GDM) [42]. They are as follows,

Low risk (blood glucose screening not routinely required)

- Member of ethnic group where GDM prevalence was low
- Age less than 25 years.
- Negative history of diabetes in the first degree relative
- Negative history of abnormal glucose metabolism
- Normal weight before pregnancy
- Normal weight at birth.

Moderate risk (blood glucose testing done at 24- 28 weeks by 1 step or 2 step procedure) :

- Member of ethnic group that had high prevalence of GDM
- Age > 25 years.
- Diabetes in the first degree relative
- Over weight before pregnancy
- High birth weight

High risk (glucose testing at the first antenatal visit / as soon as possible):

- Obesity
- Previous history of GDM
- Previous history of impaired glucose tolerance/ glycosuria
- Prior macrosomic baby
- Strong family history of type 2 DM

SCREENING OF GESTATIONAL DIABETES MELLITUS:

Screening in GDM is of utmost importance as early diagnosis can reduce both fetal and maternal complications. There has always been a debate about whether the screening should be universal or selective. The American diabetes association 4^{th} international workshop on GDM advocated that screening should be done in all intermediate and high risk women[43]. Opinions also differ regarding screening and diagnostic techniques due to differences in population risks and risk benefit ratio.

Random blood sugars , timed sample in relation to meals , serum fructosamine and HbA1c are poor screening tests[44]. Fasting plasma glucose has a high false positive rate. Women who have normal fasting and postprandial levels of glucose might show an exaggerated response to glucose challenge.

TWO STEP APPROACH:

It was recommended by the ACOG . Irrespective of the prandial state , 50 g of anhydrous glucose was given orally. Venous blood glucose was measured an hour later. This is the glucose challenge test. The test was considered positive , if the glucose level was higher than 140mg/ dl. A positive test is followed by the oral glucose tolerance test (OGTT)

The OGTT is done is done after an overnight fasting of 8 hours preceded by an unrestricted diet for 3 days (atleast 150g carbohydrates per day and unlimited physical activity). 100g anhydrous glucose solution is offered . The cutoffs are the following

Blood glucose levels	**Carpenter and Coustan criteria (in mg/dl)**	**National diabetes data group criteria (in mg/dl)**
Fasting	95	105
1 hour	180	190
2 hour	155	165
3 hour	140	145

If two or more of the values are abnormal a diagnosis of GDM is made.

Table 1: ACOG and ADA Recommendations for Diagnosing GDM[a]

	ACOG Criteria (use either)		ADA Criteria
	Carpenter and Coustan	National Diabetes Data Group	
Status of Glucose			
Fasting	95 mg/dL	105 mg/dL	92 mg/dL
1 h	180 mg/dL	190 mg/dL	180 mg/dL
2 h	155 mg/dL	165 mg/dL	153 mg/dL
3 h	140 mg/dL	145 mg/dL	N/A

[a] ACOG recommends using a 100-g OGTT and choosing one of the two threshold sets listed above (Carpenter and Coustan or National Diabetes Data Group). ADA recommends using a 75-g OGTT and the listed threshold set. One set should be chosen to use consistently in a practice setting.
ACOG: American College of Obstetrics and Gynecology; ADA: American Diabetes Association; GDM: gestational diabetes mellitus; OGTT: oral glucose tolerance test.
Source: References 1, 5.

ONE STEP APPROACH :

The HAPO trial [45] showed there was increased maternal and perinatal complications with increasing levels of glycemia even in the non diabeticrange or even in those sugar level ranges that were considered normal in pregnancy. This lead to the development of one step approach.

Here a 75g OGTT is done and plasma glucose levels are estimated after 1 and 2 hours. GDM is diagnosed if any one of the 3 values are met (IADPSG 2011)[46] .

Fasting	≥92mg/dl
1 Hour	≥180mg /dl
2 hour	≥153 mg/dl

GDM SCREENING IN INDIA :

Indians are a high risk population for the development of Diabetes with the well known fact India being the diabetic capital of world screening for GDM is of prime importance in our antenatal mothers. The DIPSI(Diabetes in pregnancy study group India) [47] guidelines is followed in India

- Universal screening is followed
- Single step procedure is followed
- Its both screening as well as diagnostic
- Irrespective of the prandial state, the pregnant women is given 75g glucose load orally and a 2hour blood glucose value is measured if the cut off is more than or equal to 140mg /dl she is branded to have GDM.

The advantages are :

- Fasting state is not required, the test can be performed at the first visit itself and repeated again in the successive trimesters.

- Both screening and diagnostic procedure
- No interference in the daily routine of women

The rationale behind the test is a normal glucose tolerant women is expected to maintain a euglycemic status despite the glucose challenge. In case of GDM the glycemic excursion exaggerates further .

FETOMATERNAL COMPLICATIONS OF GDM

GDM causes complications in both the mother and fetus.The fetus experiences complications in utero as well as in the neonatal period. Long term complications in growth and neuro development can occur in later life and there is increased predisposition to metabolic X syndrome. As far as the mother is concerned apart from the antenatal complications,there is increased predisposition to development of type 2 Diabetes in later life increasing the morbidity.

Mother	Pregnancy	Labor	Postpartum and beyond
	↑ Pre-eclampsia	↑ Induction of labor ↑ Cesarean section ↑ Operative deliveries ↑ Labor complications	↑ Recurrent GDM ↑ Type 2 diabetes
Offspring	**Congenital**	**Neonatal complications**	**Long-term outcome**
	– CNS – Cardiac Fetal programming – ↑ LGA – ↑ Macrosomia – Increased fat mass	Prematurity Perinatal asphyxia Respiratory distress Metabolic complications (hypoglycemia and hypocalcemia) Polycythemia and hyperviscosity Low iron stores Hyperbilirubinemia Cardiomyopathy	↑ Obesity ↑ Type 1 diabetes ↑ Type 2 diabetes ↑ Metabolic syndrome

MATERNAL COMPLICATIONS OF GDM

HYPERTENSIVE DISORDERS DURING PREGNANCY:

It has been found that women with GDM have an increased risk of development of preecclampsia when compared to non GDM Pregnant women. Preecclampsia is usually diagnosed with new onset hypertension and Proteinuria during 2nd half of an antenatal period. Recent ACOG Guidelines say that the presence of Proteinuria is not mandatory for the diagnosis of preecclampsia and the condition can be diagnosed even in the absence of Proteinuria in women who are hypertensive along with deranged liver function ,progressive renal insufficiency, Thrombocytopenia, Pulmonary odema, new onset cerebral or visual disturbances. A portion of this risk is due to coexisting mutual risk factors between GDM and precclampsia[48] These include obesity, positive family history, increased maternal age etc. GDM per se is an independent risk factor for the development of precclampsia,the relative risk ranging from 1.4 to 2.5[49,50,51].

Insulin Resistance in Preecclampsia

Studies have shown that women with preecclampsia are more insulin resistant prior to pregnancy as well as in first and second trimesters and in years following pregnancy when compared to normotensive pregnancy. Conditions associated with increased insulin resistance like GDM, obesity, polycystic ovary syndrome were also found to be risk factors for the development of hypertension during pregnancy[52]. Laboratory parameters in the metabolic

syndrome of insulin resistance are observed more frequently in women with hypertension during pregnancy. These parameters include

- Hyperinsulinemia
- Hyperlipidemia
- Increased levels of Plasminogen activator inhibitor – 1
- Leptin
- Tumor Necrosis factor alpha

Casey et al[49] compare 61209 pregnant women who were nondiabetic to 874 GDM women who were on medical nutrition therapy. It was found that there was an increased risk of hypertensive disorders in the GDM population (17%) when compared to non diabetic women (12%) Joffe et al[50] did the calcium for preecclampsia prevention trial. (CPEP) in 4598 nulliparous women. There was an increased risk of preecclampsia in women with GDM the relative risk being 1.67.

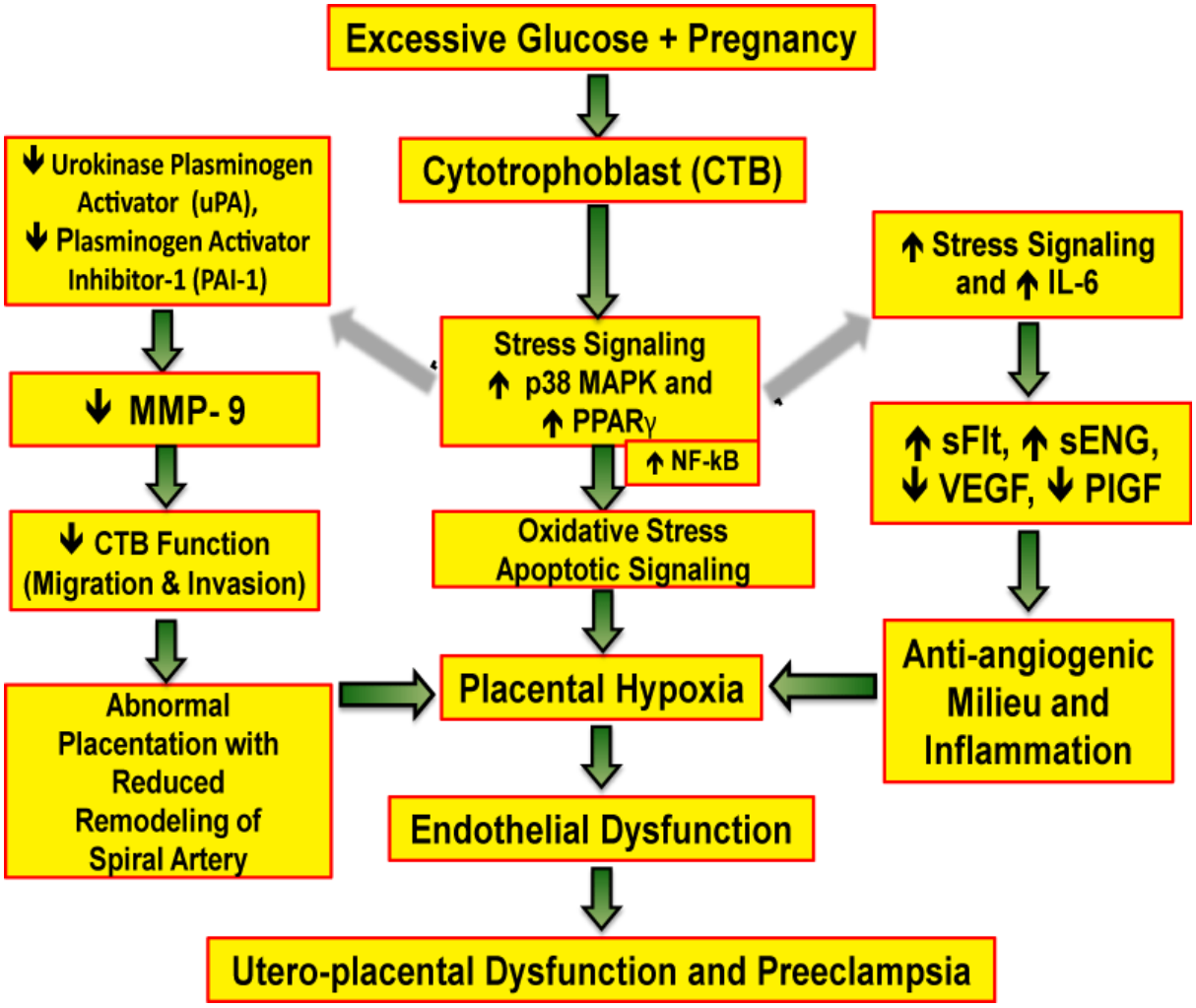
Yogevet al[51] studied 1813 GDM patients and demonstrated that preecclampsia is GDM patient is diagnosed at a younger age during first pregnancy, and in those with higher gestational weight gain. The rate of preecclampsia paralleled with the severity of GDM at diagnosis and also with the level of glycemic control. This data was strengthened by the results of HAPO study.

PLASMA GLUCOSE OF 1 SD.	THE ODDS RATIO FOR PREECCLAMPSIA
Fasting	1.21 (95% CI 1.13 to 1.29)
One hour	1.28 (95% CI 1.2 to 1.7)
Two hour	1.28 (95% CI 1.2 to 1.7)

A common etiological pathway underlies both GDM and preecclampsia. Many maladaptations to pregnancy are present in both the conditions. These include

- Endothelial dysfunction
- Angiogenic imbalance (high SFllt-1/low PGF)
- Increased oxidative stress (high free radicals / low antioxidant)
- Dyslipidemia

Chesley reported an increased risk of late onset diabetes in women with previous history of preecclampsia. When considering both pregnancy conditions, the risk of developing diabetes is moderately increased in women in who had preeclampsia alone, greatly elevated in women who had GDM alone and highest in women who had both GDM and preeclampsia.



CESAREAN SECTION AND INSTRUMENTAL DELIVERIES.

There was increased caesarean section rates and operative vaginal delivery in women with GDM. It was independent of the birth rate the caesarean section rate was approximating to 30%[53]. Tri-hospital GDM study[53] was conducted in 1996. It studied the rate of caesarean delivery in relation to birth weight & glucose tolerance. Women treated with GDM, untreated borderline GDM were cases and the controls were antenatal women without glucose

intolerance. The results demonstrated that untreated women with borderline GDM had increased rate of caesarean section (29.6%) when compared to controls. (29.2%). As did had women with treated GDM compared to controls. (odds ratio 2.1).

Even in women treated with GDM although the birth weight was normalised they remain at a higher risk of caesarean delivery of about 33%. This suggest that diagnosis of GDM lowers threshold for intervention by caesarean delivery independent of birth weight. The risk is increased nor only for caesarean deliveries but also for operative vaginal deliveries. Compared with nondiabetic women with GDM are more prone to the risk of Operative Vaginal Delivery. This risk was significantly increased by the degree of glucose intolerance (FPG > 105 mg/dl.) and maternal weight. Factors associated with the higher incidence of caesarean section in GDM include

- Age above 30 years
- Occurrence of prior caesarean deliveries
- Fetal macrosomia
- Cephalopelvic disproportion
- Unfavourable cervix for induction
- Fetal distress
- Risk of intrauterine death
- Gestational age < 37 weeks or > 40 more weeks

POLYHYDRAMNIOS

Polyhydramnios is defined as pathological increase of amniotic fluid volume in pregnancy. It is diagnosed ultrasonographically when amniotic fluid index (AFI) > 25 cms. Or when single vertical pocket is > 8 cms. It can be due to various causes, common most include fetal anomalies and gestational diabetes mellitus. There exists a dynamic equilibrium between production and resorption of amniotic fluid. Fluid levels are influenced by fetal urination and fetal lung liquid production. Polyhydramnios complicates 5 to 25% of diabetic pregnancy. The various underlying mechanism includes

- Fetal hyperglycemia causing fetal polyuria resulting in increased osmotic diuresis

- Placentomegaly increasing the surface area of the placenta leading to increased amniotic fluid volume
- Associated congenital anomalies and metabolic derangements

The prevalence of polyhydamnios is 18.8%. It can be an indicator of diabetogenic fetopathy. The various complications of polyhyramnios include

- maternal dyspnea
- increased exhaustion
- premature rupture of membrane
- preterm labour
- fetal malposition
- umblical cord collapse
- uterine atony
- abruptioplacentae
- abdominal discomfort due to overdistended uterus

PRETERM LABOUR

It is defined as delivery before 37 weeks of gestation. It is one of the maternal complications of GDM. However it is less common when compared to other adverse outcome. In the HAPO study 6.9% of the participants experienced preterm delivery both spontaneous and induced. Out of which 9.6% of infants were LGA where 8% underwent intensive neonatal care[54]. It was also

observed that preterm labour had minimal association with fasting glucose levels as well as maternal blood pressure. The association of pre term labour with GDM can be attributed to coexisting preeclampsia, placental abruption, recurrent urinary tract infection, polyhydramnios. It should also be noted that 3/4ths of preterms births are not associated with GDM and are spontaneous.

URINARY TRACT INFECTION

Pregnancy causes hormonal and mechanical changes that increase the risk of urinary stasis and vesicoureteral reflux. These changes along with short urethra increase the frequency of urinary tract infection (UTI) in pregnant women.

UTI is defined as presence of atleast 1.0 lakh organism / ml. of urine in asymptomatic patients or > 100 organisms / ml. of urine with accompanying pyuria in symptomatics supported by a positive culture of uropathogen.

Infections are mainly due to ascending colonisation of existing vaginal perineal and faecal flora. Enlarging uterus causes urinary retention and stasis due to progesterone induced, ureteral smooth muscle relaxation. Glycosuria and increased levels of urinary aminoacids during pregnancyt are additional factors that lead to UTI [55] When compared to nondiabetic women, women with GDM have a higher incidence of UTI (Prevalence in nondiabetic – 3 to 10%, GDM women 27.6%) This UTI can be either asymptomatic bacterurea or

acutecystitis, less commonly acute pyelonephritis. Apart from anatomical and physiological changes in renal tract during pregnancy in GDM

- ✓ there is suppression of immune system enhancing the progress of the condition.
- ✓ Decreased antibacterial activity of urine as a result of dilution of inhibitory substances such as urea.
- ✓ Defect in polymorphonuclear leukocyte function due to hyperglycemia and increased adhesive capacity of bladder epithelium

It was found that GDM was not a risk factor for postpartum UTI because there was no significant difference in the incidence of the disease in GDM women and normal women. 20 to40 % of pregnant bacteriuric women whether diabetic or not develop acute pyelonephritis during pregnancy if treatment is not provided. Furthermore recurrent UTI increases the risk of preterm labour,chorioamnionitis, premature rupture of membranes and intrauterine and neonatal sepsis.

INCREASED RISK OF POSTPARTUM TYPE T2DM

Abnormal Carbohydrate tolerance following pregnancy with GDM was evaluated in numerous studies. After 28 years of follow up of the original cohort from O'Sullivan's work that determined OGTT cut offs for GDM nearly half of the women with GDM had T2DM in later life. Kjos et al[22] performed 75

gm. OGTT 5 to 8 weeks after delivery in 246 women with GDM and found 19% had abnormal OGTT, out of which 10% had impaired glucose tolerance and 9% had T2DM. Sivaraman et al[56] reported the risk of developing diabetes was 6.9% at 5 years. And 21.1% at 10 yrs. following initial diagnosis of GDM.Chodick et al found that the risk of T2DM among women with prior GDM was 15.7% upto 10 yrs. of follow up. O'Sullivan consolidated available data and reported a large variance in the risk of postpartum T2DM ranging from 6% to 60% when they were evaluated from 6 weeks after deliver to 10 yrs. later. The cumulative incidence of T2DM increased remarkably in the first five years of delivery and plateaued after 10 years.

Risk factors for developing T2DM include[58]:

- Ethnicity (e.g. African-American, Latino, Native American, Asian American, Pacific Islander)
- High Parity
- Age at delivery ≥ 33-35 years
- Family history of diabetes
- Duration of follow up after pregnancy
- Early Diagnosis of gestational diabetes mellitus (< 22-24 weeks)
- Testing modality for diagnosing diabetes (e.g. Oral glucose tolerance test, Fasting plasma glucose, Random plasma glucose or Hemoglobin A1C)

Severity of gestational diabetes mellitus

- Degree of hyperglycemia in pregnancy and immediately postpartum
- Number of abnormal Oral glucose tolerance test values
- Total area under the diagnostic Oral glucose tolerance test
- Level of fasting blood glucose on the Oral glucose tolerance test
- Elevated fasting glucose level during pregnancy
- Need for pharmacological therapy to achieve glycemic control

Lifestyle parameters:

- Consumption of dietary fat
- Limited physical activity
- Smoking

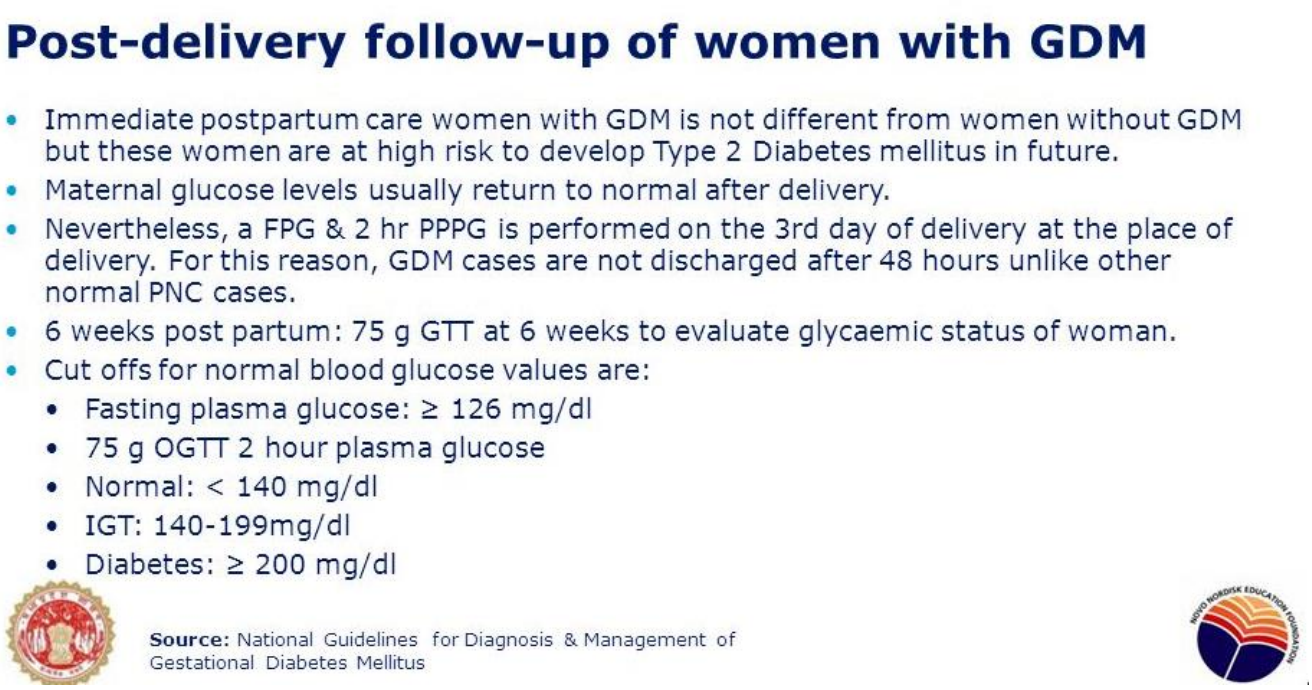
Maternal Weight:

- Gestational weight gain
- Pre-pregnancy weight and Body Mass Index
- Postpartum weight retention

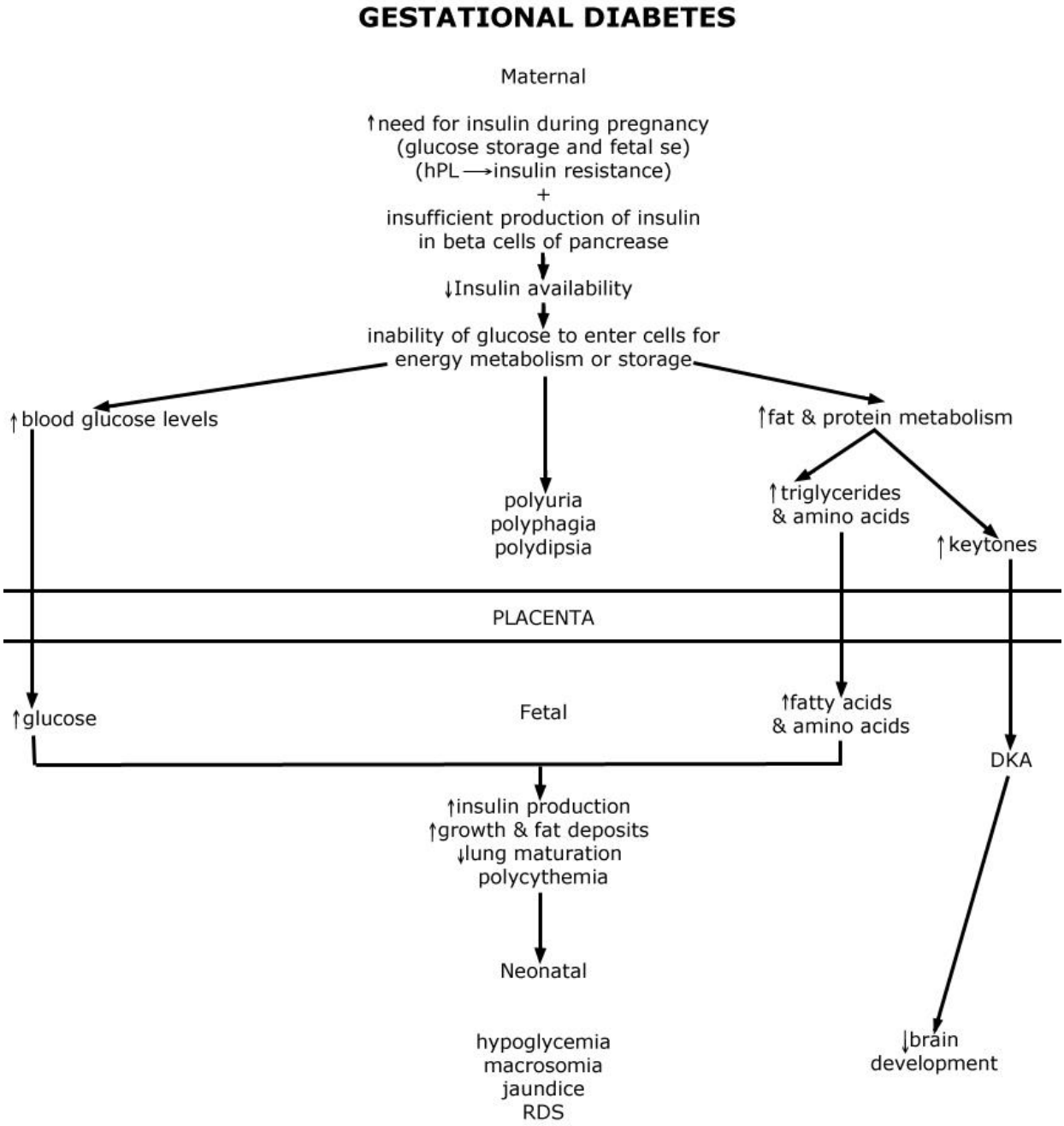
Metabolic syndrome parameters at early postpartum:

- High-density lipoprotein cholesterol > 50mg/dL
- Waist circumference of 88cm or higher

ACOG recommends postpartum in all GDM patients at 6-12 weeks after delivery using a 75 gm OGTT[59]. The screening is carried out annually for three years is values are normal and once in three years for the successive years lifelong.



FETAL COMPLICATIONS



Gestational Diabetes Mellitus is associated with an array fetal complications and the neonate is also affected in the long run. The various complications in the fetus includes:

- Congenital Malformation
- Unexplained Intrauterine Demise
- Still Born
- Increased Birth Weight / Macrosomia
- Shoulder dystocia & Birth Injuries
- Fetal Asphyxia
- Perinatal Death
- Neonatal Hypoglycaemia
- Hyper Bilirubinemia
- Transient Tachypnea
- Hypocalcemia
- Cardiomyopathy
- Polycythemia

CONGENITAL MALFORMATIONS

The incidence of congenital malformations is higher in pregestational diabetes when compared to gestational diabetes mellitus. High risk of spontaneous abortion as well as malformation of the fetus occurred first trimester of pregnancy especially when the glycemic control was poor. The frequency of birth defect is 3 to five foldincreased when compared with the general population. SHAEFER-Graf et al[60], in a review of 4180 pregnancies complicated by GDM reported that congenital anomalies in the offspring

affected the same organ systems described in pregnancies complicated by IDDM – Insulin Dependent Diabetes Mellitus. However most other reports have conflicting findings as GDM most commonly occurred after the period of organogenesis and have minimal effect on teratogenicity.

The Swedish Health Registry Study covered 1.2 million birth between 1997 and 2007 and identified 3864 infants were born to women pre-existing diabetes and 8688 infants born to women with GDM[61]. The total malformation rate in the first group was 9.5% and the second group 5.7%. Although the risk of anomalies in the GDM group is lower when compared to the pre GDM group, it has a higher incidence when compared to the general population. Martinez-Frias et al[62]analysed 19577 consecutive infants with malformations for unknown cause and compared those born to mothers with GDM with those of non diabetic mothers. Their findings indicated GDM is a significant risk factor for holoprosencephaly, upper / lower limb anomalies, spine anomalies, renal and urinary system anomalies. However owing the heterogenous nature of GDM which includes previously unrecognised and newly diagnosed non Insulin Dependent Diabetes Mellitus they could not rule out the possibility that the teratogenic effect is related to latent NIDDM. Nevertheless pregnancies complicated by GDM should be considered at risk for congenital anomalies.

The various factors responsible for these malformations include hyperglycemia which might have teratogenic effect during the organogenesis

period. Free radical injury causing embryopathy and other metabolic derangement like deficiency of arachidonic acid and myoinositol inhibition of cellular uptake of dehydroascorbic acid and increased nonenzymatic glycosylation of embryonic proteins, abnormal levels of Trace metals and decreased catalyse activity.

Unexplained Intrauterine Demise

Gestational diabetes mellitus has a higher risk of facing the disaster of Unexplained Interauterine Demise especially during the third trimester towards term. Careful monitoring of the fetal wellbeing especially as pregnancy reaches term is of significant importance. Antepartum fetal surveillance should be started at 32 weeks of gestation. Women with uncontrolled gestational diabetes need to be hospitalised and glycemic control should be achieved. GDM women on insulin are terminated at 38 weeks of gestation and are not allowed to reach 40 weeks as the chance of intrauterine device increases with advancing gestational age. The possible cause of fetal demise includes fetal hypoxia which could be attributed to the following reasons:

- The glycosylated haemoglobin had decreased oxygen carrying capacity and causes leftward shift of haemoglobin and oxygen dissociation curve.
- Decreased nutrient and oxygen transport to the fetus owing to placental villous odema and vasculopathy.

- Associated preeclampsia causing vaso constriction and thereby decreased nutrient supply.

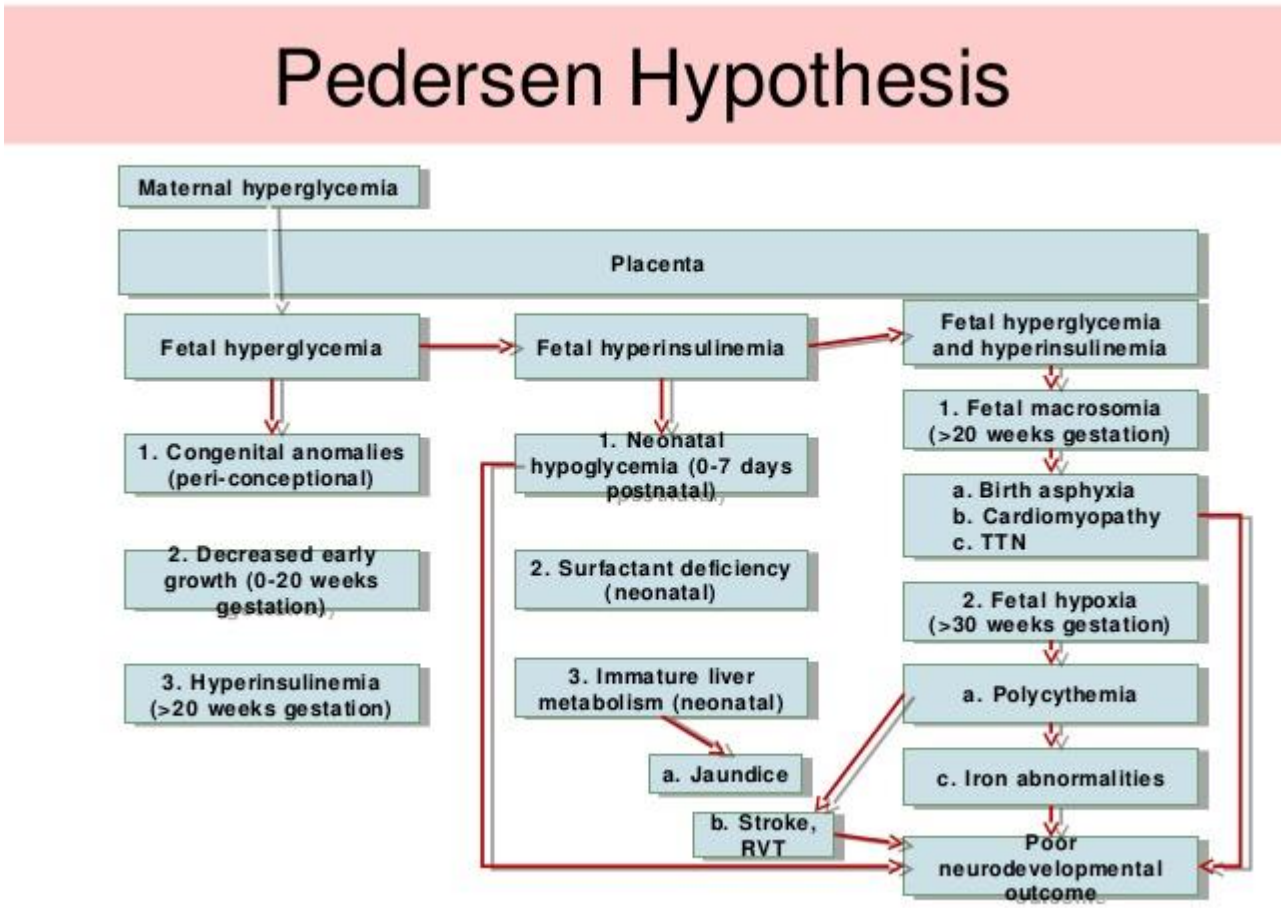
Rackham et al found that there was a curvilinear relationship between HbA1c and fetal weight and uteroplacental blood flow and consequent fetal hypoxia.

Increased Birth Weight / Macrosomia

Macrosomia is defined as birth weight of 4000 gm. and above regardless of the Gestational age[63]. Gestational diabetes mellitus is an independent risk factor for fetal macrosomia and large for gestational age babies. 50% of GDM pregnancies can be complicated by fetal macrosomia. The incidence of macrosomia in those with untreated GDM is high as 20% compared to only 6% in those who were appropriately treated. The incidence of macrosomia was only 2% in those without diabetes.

The macrosomic fetus has thick skinfold higher body fat percentage increased shoulder circumference, small head to abdomen circumference ratio leading to altered body shape and composition. This alteration in the body shape attributes to higher risk of shoulder dystocia, birth trauma, post partumhaemorrhage and ceasarian deliveries. Risk of PPH in macrosomia is doubled.

Human fetal growth is influenced by multiple factors like parental genome placental sufficiency, meternal nutrition and lifestyle. Pathological variation in birth weight is associated with changes in fetal production and action of insulin. The increased body mass of the fetus in GDM results primarily from fetal hyper insulinemia as result of maternal hyper glycemia. These foetuses have Beta cell hyperplasia. This was explained by Professor Jorgen Pederson [64] in his hypothesis. He found that maternal hyperglycemia led to fetal hyperglycemia which stimulated pancreatic beta cell hyperplasia in the fetus and led to fetal hyperinsulinemia. Insulin and insulin like growth factors exert an anabolic effect due to their mitogenic and antiapoptotic effects on cell differentiation thereby causing increase in skeletal growth and fat deposition. The pattern of overgrowth is complicated consisting of visceromegaly of liver, heart, lungs, adrenals and subcutaneous adiposity.



Effect of insulin like growth factor on islets.

Gene	Phenotype
IGF2	Increased islet size
IRS1	Increased islet cell mass
Insulin	Increased islet size
IRS – Insulin Receptor Substrate	
IGF – Insulin like growth factor	

The Pedersen Hypothesis has been extended. It states that intrinsic fetal pancreatic beta cell hyperplasia pulls glucose across the placenta and assists in glycemic control of the mother. The initial increase in fetal size gives rise to hypoxemia and limitation in fetal oxygen availability alters differential tissue utilisation of glucose leading to alphaglycerophosphate synthesis in fetal adipocyte thereby increasing further adiposity. Thus it can be concluded that optimal correction of hyperglycemia in the mother can lead to significant alteration in the fetal adiposity thereby preventing macrosomia and it's related consequences.

Delayed fetal lung maturity

The diabetic pregnancy,especially poorly controlled GDM is an important risk factor for the development of respiratory distress syndrome. Planning of both therapy and delivery is very critical and crucial in improving the offspring outcome. Robert and Neff found that risk of RDS was six times higher in GDM women when compared to normal women[65]. Hyperglycemia and hyperbilirubinemia are involved in delayed pulmonary maturation which influences pulmonary surfactant biosynthesis. Carlson and Smith showed that insulin blocks cortisol action at the level of fibroblasts by decreasing the production of fibroblast pneumocytic factor. It also interefered with the timing of corticoid induced maturation in the fetus. The various tests for fetal lung maturity include:

- Shake test
- Planimetric LS ratio
- Stochiometric Ls ratio
- Amniotic Fluid Phosphatidyl glycerol
- Lamellar body count

  Strict blood glucose control is essential in pregnancy and decision to deliver should be made when the fetal lung maturity has been ascertained.

Shoulder dystocia & Birth Injuries

Shoulder dystocia is diagnosed when additional maneuvers are required to deliver the shoulder if traction on the fetal head does not suffice. In the HAPO study,shoulder dystocia was one of the least common outcomes only 1.3% of women being affected. It increases the risk of birth trauma and can cause fetal asphyxia if neglected or late diagnosed. The condition occurs mainly in macrosomic fetus where there is increased fat on the shoulder. Increased odds of shoulder dystocia has been observed in women with glucose intolerance duting antenatal period. It is mainly due to abnormal anthropometric diameters like truncal obesity,large shoulder diameter as well as increased weight of the mother.

The ACHIOS trial (Australian Carbohydrate Intolerance Study in Pregnant women) observed whether treatment gestational diabetes reduced the

perinatal complications. Primary outcomes included shoulder dystocia,bonefracture,nerve palsy and it was found that the risk of serious perinatal complications were lower in infants of the intervention group when compared to the routine care group. A positive relationship was found between maternal hyperglycemia in the fasting state and the risk of shoulder dystocia.1 mmol rise in fasting OGTT leads to a relative risk of 2.09. Dystocia occurred mostly in births requiring operative vaginal delivery.

Brachial plexus injuries is one of the most important complication of this condition present in 4-16%. It was found to be independent and unrelated to the operator experience. Majority reolved without permanent disability while permanent dysfunction was seen in less then 10%..

PERINATAL MORTALITY

The perinatal mortality is increased in GDM women when compared to the normal population and it has a direct relationship with the stringency of glycemic control. This could be due to various causes like congenital anomalies, unexplained still births,intrapartumasphyxia,unexplained stillbirths, respiratory distress syndrome,neonatal sepsis etc.

SHORT TERM IMPLICATIONS IN THE NEONATE

NEONATAL HYPOGLYCEMIA

Most dreaded complication in the neonate of the GDMmother. It is defined as plasma glucose concentration<40 mg % or serum glucose concentration <45 mg %. Codero et al[66]observed that neonatal hypoglycemia rate in GDM babies was 25%. The most accepted definition of neonatal hypoglycemia is the Pedersen hypothesis which has been already discussed. The hypothesis was further extended by Freinkel,who examined the role of other nutrients that provided substrate for the fetus. He introduced the concept of pregnancy as a "tissue culture experience"proposing that the placenta and the fetus develop in an incubation medium derived from maternal fuels[1]. As all this constituents are regulated by maternal insulin,any disturbance in its supply or action can lead to hyperinsulinemia. Other factors like defective counter regulation by catecholamines or glucagon lead to increased glucose tolerance and decreased glucose production. Early diagnosis and prompt management of the condition is essential to prevent late consequences of acute neurological injury and permanent neurological sequelae.

NEONATAL HYPOCALCEMIA

It is defined as serum calcium level <8 mg % in term infants and <7 mg% in preterm infants. The severity of the condition is linked to the severity of

maternal hyperglycemia,the reported rate being 10-20% in GDM babies[67]. Hypocalcemia could be due to the following reasons:

- Parathyroid hormone concentrations are lower in IDM than infants of non diabetic mothers during the first four days of life.
- Glycosuria induced loss of maternal urinary magnesium leads to hypomagnesemia that inhibits PTH secretion leads to hypocalcemia.
- Persistently high levels of Calcitonin in IDM leading to hypocalcemia

  If promptly treated it has a good prognosis even in those with convulsions.

RESPIRATORY DISTRESS SYNDROME

It is constellation of clinical,radiological and laboratory findings due to deficient surfactant production leading to decreases lung compliance and hypoxia. Robert et al[68] found a 5-6 fold rise in RDS in IDM than those of non diabetic mothers. The RDS incidence dropped significantly from 31% to 3% with introduction of strict glucose control during pregnancy and delivery at term or near to term.

- Fetal hyperinsulinemia blocks normal enzyme inducing action of cortisol on fetal type 2 pneumocyte production of surfactant due to inhibited production of phosphatidyl choline and fibroblast pneumocyte factor.

- Insulin converts glycerol –b-phosphate to pyruvate and acetyl coA decreasing its availability for phospholipid synthesis[69].
- Insulin interferes with the conversion of phosphatidic acid to phosphatidyl glycerol which has a stabising effect on surfactant.

  As phosphatidyl glycerol signal final maturation of surfactant production,once it appears the infants of GDM and Pre GDM mothers can be delivered. If elective delivery before 38 weeks of gestation in planned fetal lung maturity should be ascertained.

Other early neonatal complications include:

- Polycythemia
- Hyperbilirubinemia
- Thrombocytopenia
- Ventricular septal hypertrophy

GROWTH AND NEURODEVELOPMENT OF CHLDREN BORN TO GDM MOTHERS

Children born to diabetic mothers are often macrosomic,especialy when maternal hypergylcemia occurs during the second and third trimesters. On follow up,themacrosomic children tend to normalise their weight and height within the first year of life but there is a stronger tendency for increased weight

gain and development of metabolic X syndrome in later life[70]. Children born SGA tend to be smaller for atleast first several years of life.

If the sugars are well controlled in pregnancy,the intellectual function of the offspring is usualy within normal limits. However fine and gross motor abilities,attention span and activity levels are impaired among those born to GDM mothers when matched with the controls[71]. In some studies the differences compared to controls are larger in children between the ages of 5-8 and these differences are reduced in older children. It is possible that the metabolic abnormalities during pregnancy delay brain maturation and therefore fine neurological functions are impaired at an young age.

These results emphasise the importance of good glycemic control during the antenatal period thereby preventing the diasatrous effects of these long term complications in their children.

MATERIALS AND METHODS

STUDY DESIGN

Prospective observational study

PERIOD OF STUDY

One year (October 2016-September 2017)

Purpose of the study:

- The prevalence of diabetes mellitus (DM) is increasing worldwide and more in developing countries including India.
- As women with gestational diabetes mellitus (GDM) and their children are at increased risk of developing diabetes mellitus in future, special attention should be paid to this population especially in developing countries.
- Early detection and prompt management will help to decrease the maternal and fetal morbidity and prevent long term complications

All patients attending the Antenatal OPD at Chengalpattu medical college hospital were offered a 75g GCT. Those who had GCT values more than 140mg/dl were included in the study.Study involves 400 patients diagnosed with GDM irrespective of the period of gestation. Height, weight, and blood pressure were measured at everyvisit. Through proper

history taking, clinical examination and lab investigations glycemic control was achieved on medical nutrition therapy or insulin and these patients are followed up from antenatal period till six weeks postpartum. Fetomaternalcomplications , perinatal outcome ,the number of patients developing glucose intolerance postpartum (diagnosed by 75 g 0GTT) are evaluated during the study period

Inclusion criteria

- Patients diagnosed with gestational diabetes mellitus (by 75g OGCT according to DIPSI guidelines)
- Singleton pregnancy

Exclusion criteria:

- Chronic hypertension
- Patients with pregestational diabetes mellitus
- Patients on medications that can alter the glucose metabolism like steroids,antipyschotics,diuretics ,oral contraceptive pills,beta blockers
- Patients with abnormal thyroid profile
- Cushings syndrome
- Chronic medical illness
- Autoimmune disease
- Multiple pregnancy

INVESTIGATIONS

ANTENATAL:

- Hemoglobin,Platelet count
- Urine routine,urine culture sensitivity
- Serum urea,creatinine,electrolytes
- Liver enzymes
- 75g Oral Glucose challenge test
- Fasting and Postprandial blood glucose
- Ultrasonography

POSTPARTUM:

- 75g Oral Glucose Tolerance test
- Fasting and Postprandial blood glucose

PARAMETER	NORMAL VALUES
Hemoglobin	12-16 g/dl
Urine culture sensitivity	$< 10^5$ organism/ml urine
Platelet count	1.5-4 lakhs/cu.mm
Total Bilirubin	0.2-1 mg/dl
Indirect Bilirubin	0.1-1.0 mg/dl
Direct Bilirubin	0-0.2 mg/dl
AST/SGOT	5-43 U/L
ALT/SGPT	5-56 U/L
LDH	<400 U/L
Serum uric acid	3-7 mg/dl
Blood urea	<20 mg/dl
Creatinine	0.7-0.9 mg/dl
GCT	<140 mg/dl
FBS	<92 mg/dl
PPBS	<140mg/dl

OUTCOMES ANALYSED

1) Primary outcome

- Maternal outcome-mode of delivery
- Fetal outcome- Birth condition
    - Birth weight
    - Birth trauma
    - Congenital anomalies

2) Otheroutcomes

- ANTEPARTUM COMPLICATIONS:
    - Pre eclampsia
    - Urinary tract infection
    - Preterm labour
    - Premature rupture of membranes
- Postpartum: Incidence of overt diabetes mellitus/glucose intolerance

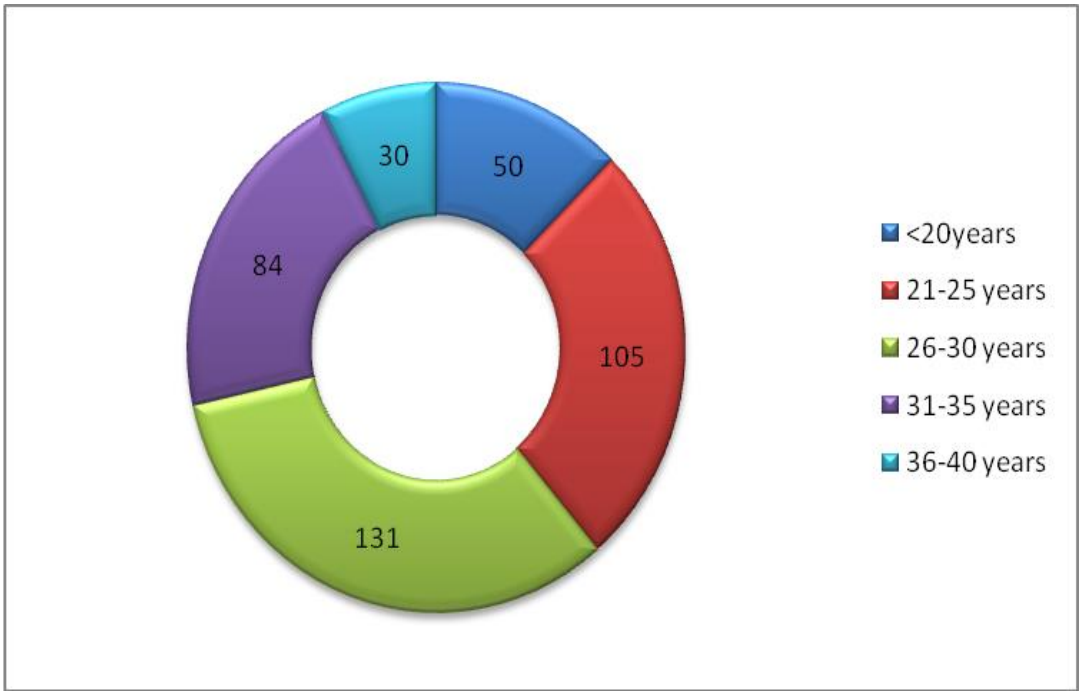
STATSTICAL ANALYSIS

- Primary data was entered in MS Excel and analyzed using SPSS 20v.
- The results were presented in terms of tables and graphs.
- The descriptive statistics frequency and percentage were calculated.
- The association between the categorical variables was analyzed by chi square test with 5% level of significance.

RESULTS AND ANALYSIS

1. AGE DISTRIBUTION

Age	Frequency	Percent
≤20years	50	12.4
21-25 years	105	26.3
26-30 years	131	32.7
31-35 years	84	21.1
36-40 years	30	7.5
Total	400	100

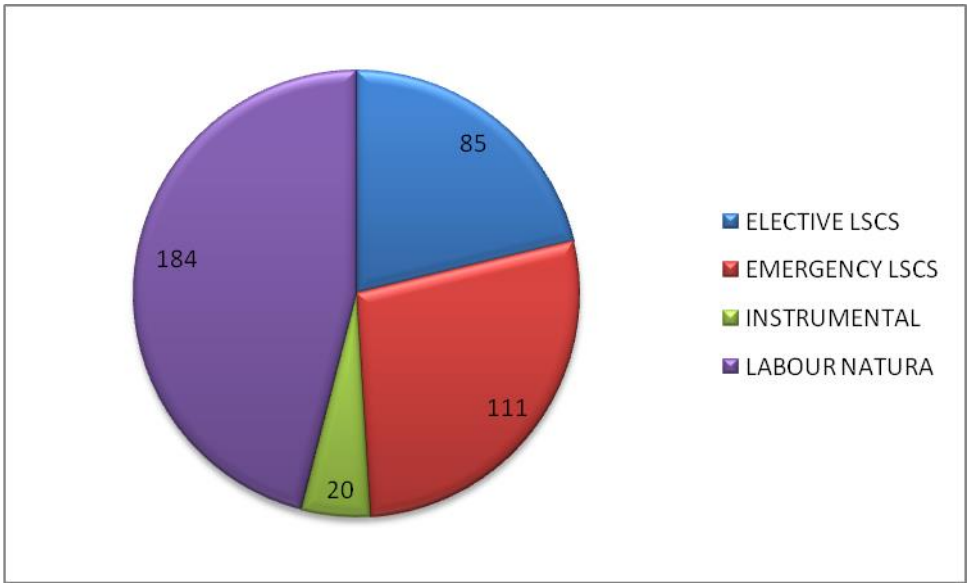


In this study,the maximum population of GDM patients came under the age group 26-30 years(32.7%).GDM in teenage pregnancy was encountered in

12.4% of the study population. The elderly gravidas covered 7.5%. In this study,the occurrence of GDM was lesser in the extremes of age group. The lower incidence in the elderly could be probably because these mothers would have had established pregestational diabetes and therefore did not meet the inclusion criteria.

2. MODE OF DELIVERY

Mode of Delivery	**Frequency**	**Percent**
LSCS	196	49
ELECTIVE LSCS	85	21.2
EMERGENCY LSCS	111	27.8
INSTRUMENTAL	20	5
LABOUR NATURALIS	184	46
Total	**400**	**100**



Majority of the study population delivered via lower segment caesarean section (49%) out of which 21.2% had elective LSCS and 27.8 % had emergency LSCS. The most common indications for emergency LSCS were the following in order

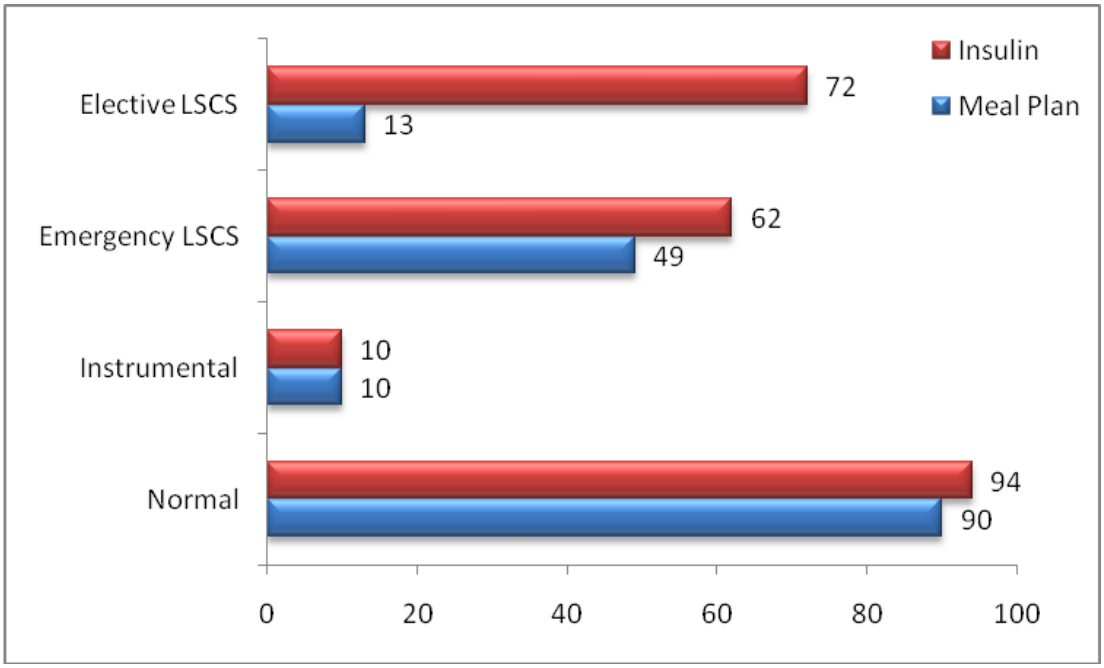
- Failed induction – 65.3 %
- Meconium stained liquor – 15.2%
- Fetal distress- 15.5%
- Cephalopelvic disproportion in labour -14%

The indications for elective LSCS included

- Macrosomia
- Repeat LSCS

51% of the study population delivered vaginally out of which 46% delivered via labournaturalis and 5 % via instrumental delivery. The most common indication for instrumental delivery was large baby with birth weight more than 3.5kg .

Mode of Delivery	**Meal Plan**	**Insulin**	**Total**	**Chi Sq**	**P**
Normal	90	94	184	29.18	**0.001**
Instrumental	10	10	20		
Emergency LSCS	49	62	111		
Elective LSCS	13	72	85		
Total	**162**	**238**	**400**		

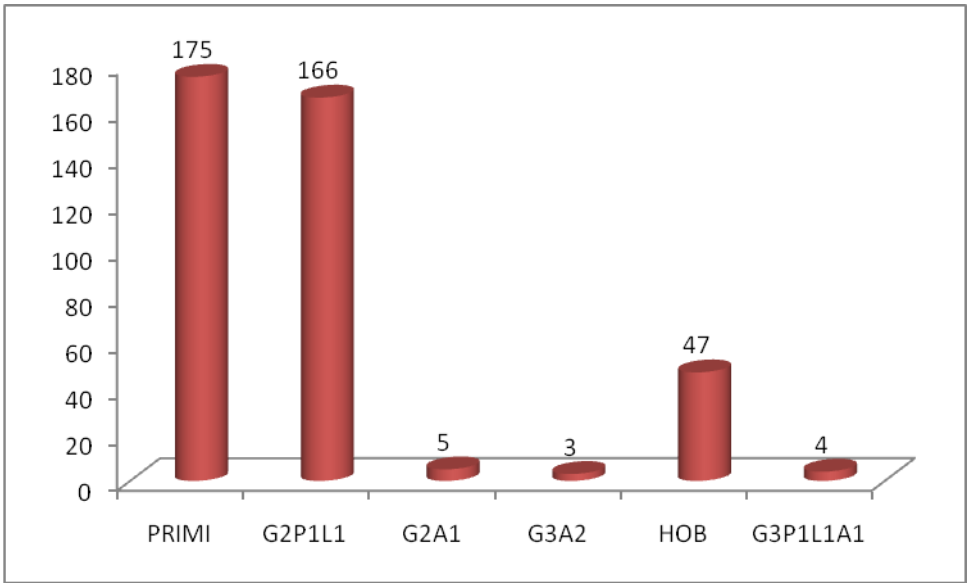


It was observed that in the study population,the Caesarean section rate was higher in those women who were on insulin when compared to those on meal plan(p value=0.001) which was statisticaly significant.

Among the 184 women who delivered via labour naturalis,94 of them were on insulin and 90 of them on meal plan. Equal proportion was also observed in the instrumental delivery(n=10 in both the groups)

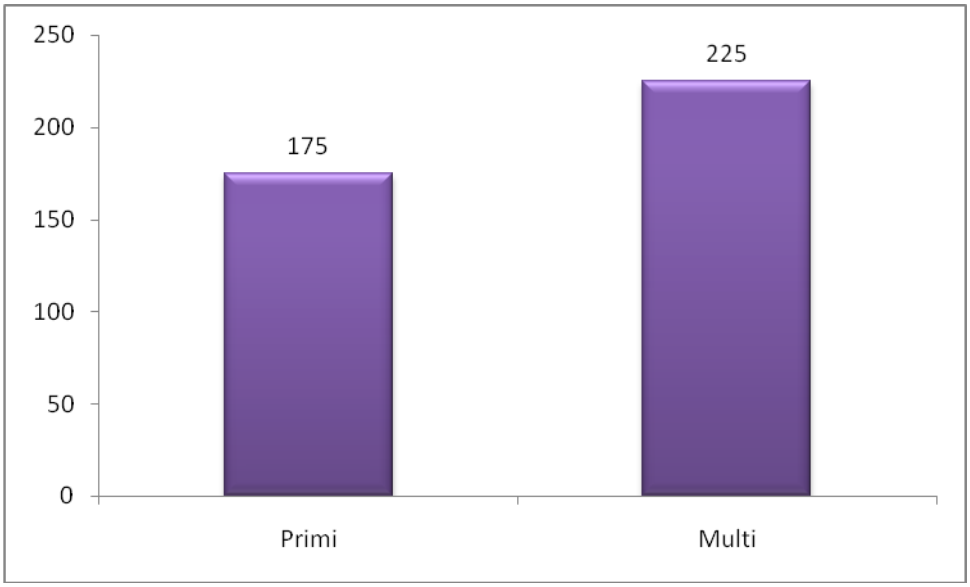
3. PARITY

PARITY	**Frequency**	**Percent**
PRIMI	175	43.8
G2P1L1	166	41.5
G2A1	5	1.2
G3A2	3	0.8
HOB	47	11.7
G3P1L1A1	4	1
Total	**400**	**100**



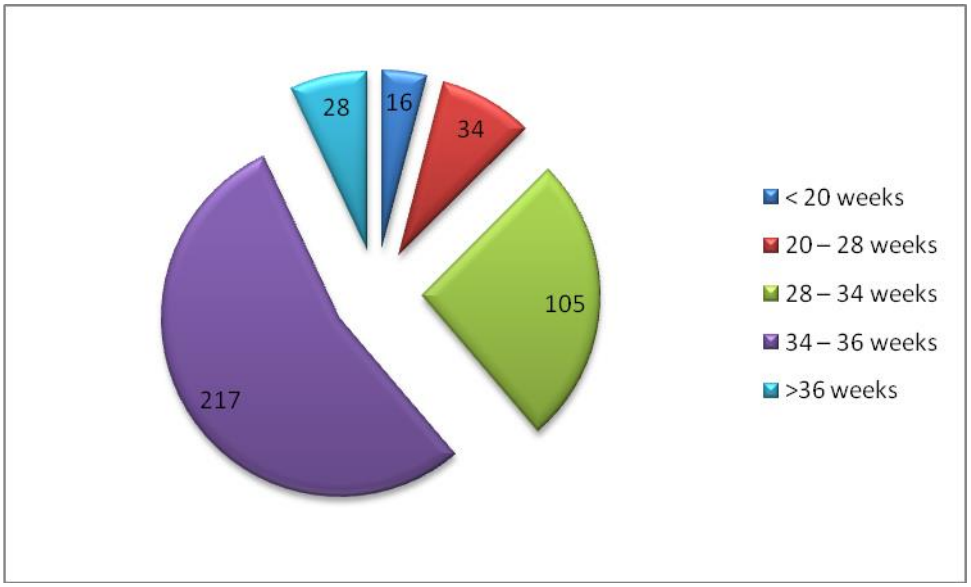
Out of 400 women , 175 women were primi gravida (43.8%). 166 women were 2nd gravidas with a previous live child (41.5%). Higher order births constituted 11.7 % . In these two groups 80% had prior uncomplicated pregnancy and 20 % had prior history of GDM, out of which 12 % had normal OGTT value in the post partum period and the remaining 8 % did not turn up for the follow up. Women with previous history of abortion constituted 2 % and in most of these women the cause of previous abortion was unknown .

	Frequency	**Percent**
Primi	175	43.8
Multi	225	56.2
Total	**400**	**100**



4. GESTATIONAL AGE AT DIAGNOSIS

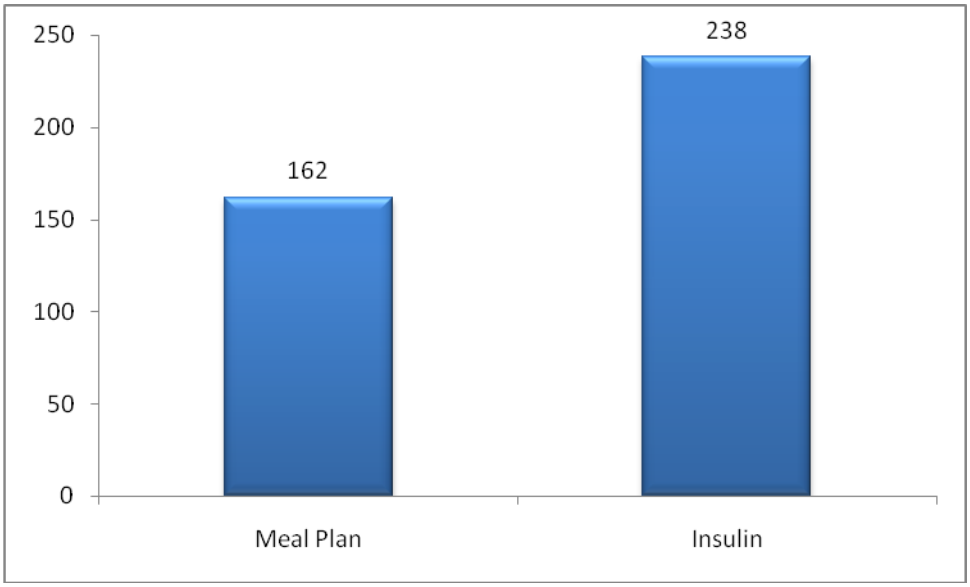
Gestational Age	Frequency	Percent
< 20 weeks	16	4
20 – 28 weeks	34	8.5
28 – 34 weeks	105	26.2
34 – 36 weeks	217	54.2
>36 weeks	28	7
Total	**400**	**100**



The maximum occurrence of GDM was between 34 – 36 weeks of gestation (54.2%) , which can be attributed to the fact that insulin resistance was maximum during the last 3 months of pregnancy that was observed in the previous studies. 26.2 % of GDM occurred between 28 – 34 weeks of gestation. The occurrence was 8.5 % in gestation between 20- 28 weeks and 7 % in gestation age more than 36 weeks . Only 4 % of the study population were diagnosed with GDM at gestational age less than 20 weeks.

5. TREATMENT PLAN

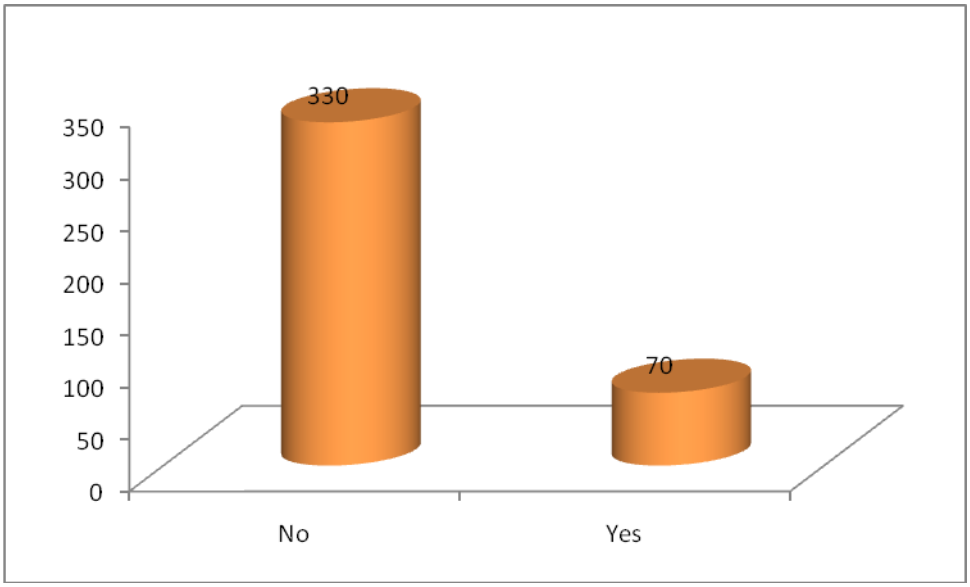
TREATMENT	**Frequency**	**Percent**
Meal Plan	162	40.5
Insulin	238	59.5
Total	**400**	**100**



In this study, 238 out of 400 women were started on insulin (59.5%) and the remaining 162(40.5%) women had their glycemic control achieved with meal plan alone.

6. POLYHYDRAMNIOS

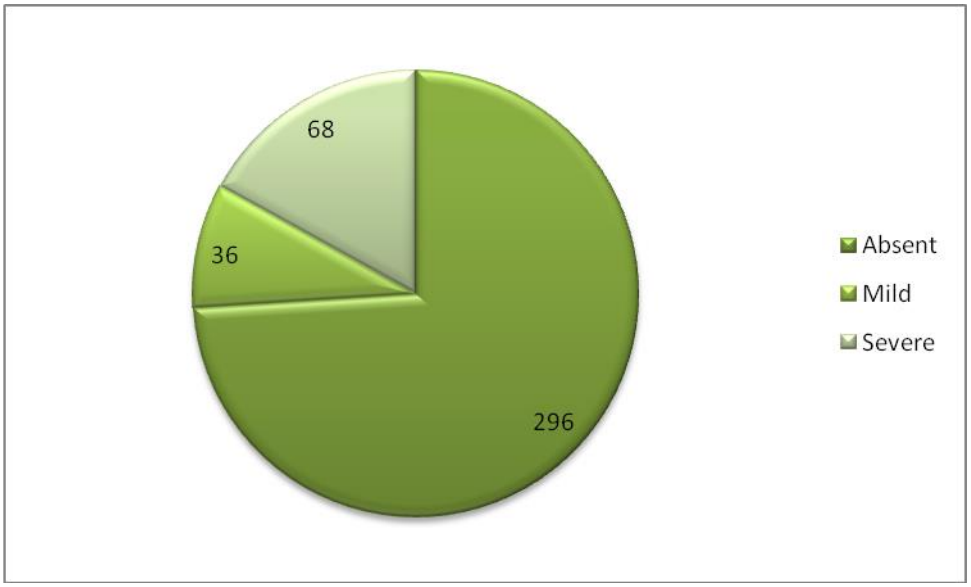
POLYHYDRAMNIOS	**Frequency**	**Percent**
No	330	82.5
Yes	70	17.5
Total	**400**	**100**



In this study, out of 400 women, 70 women had polyhydramnios (17.5%) .In these 70 women 44 women were on insulin and 26 women were on meal plan, 32 % of them underwent preterm labour. Out of these 70, 38 women had isolated polyhydramnios and the remaining 32 had other associated maternal complications.

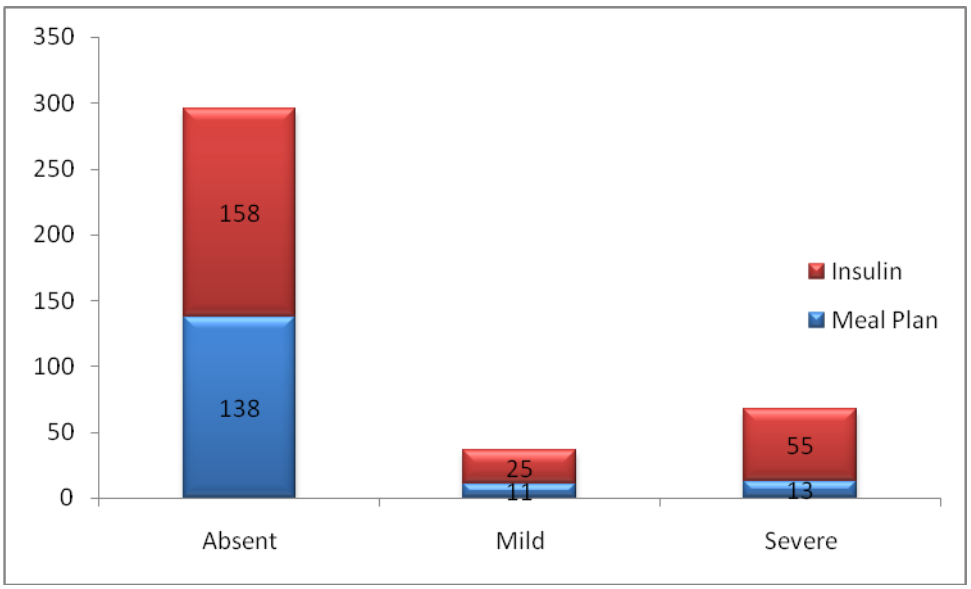
7. PRE ECLAMPSIA:

Pre eclampsia	**Frequency**	**Percent**
Absent	296	74
Mild	36	9
Severe	68	17
Total	**400**	**100**



Pre eclampsia was seen in 26 % of the study population (n = 104). Severe preeclampsia was seen in 17 % (n = 68) and mild pre eclampsia was seen in 9% (n=36). Among those who had severe preeclampsia 80% of them were on insulin (n=55) and the remaining 20 % were on meal plan (n =13). Among those who had mild preclampsia 70 % of them were on insulin (n-25) and the remaining 30 % were on meal plan (n=11).

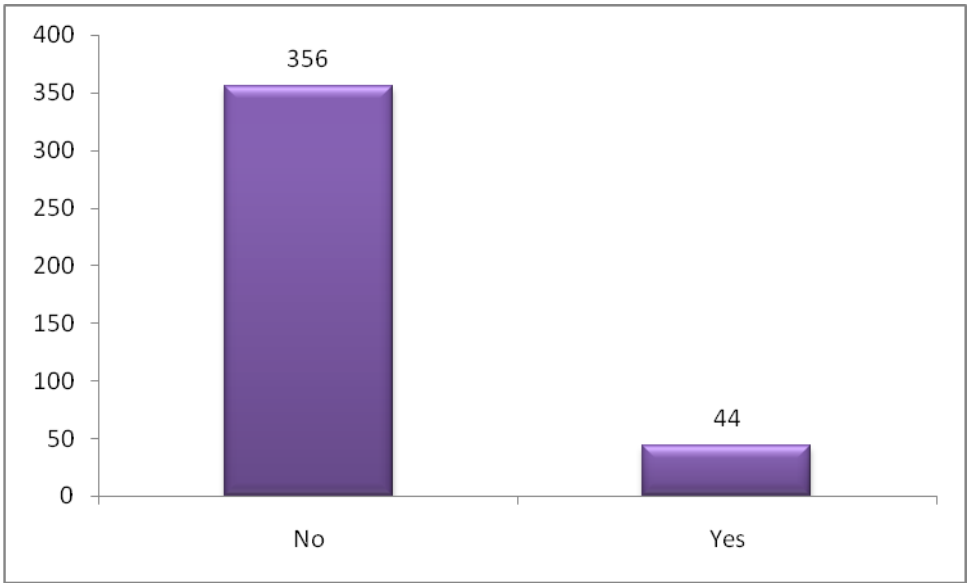
Preeclampsia	**Meal Plan**	**Insulin**	**Total**	**Chi Sq**	**P**
Absent	138	158	296	**18.98**	**0.001**
Mild	11	25	36		
Severe	13	55	68		
Total	**162**	**238**	**400**		



Out of the 400 GDM women 104 had pre eclampsia(36 women had severe pre eclampsia and 68 women had mild pre eclampsia). The incidence of pre eclampsia was more in those women who were treated with insulin than those women whose glycemic control was achieved with meal plan alone and the p value was statistically significant(p value=0.03 chi sq=18.98). This can be explained by the theory that insulin resistance also plays a role in pathogenesis of pre eclampsia and the two conditions share common factors contributing to the pathogenesis.

8. UTI

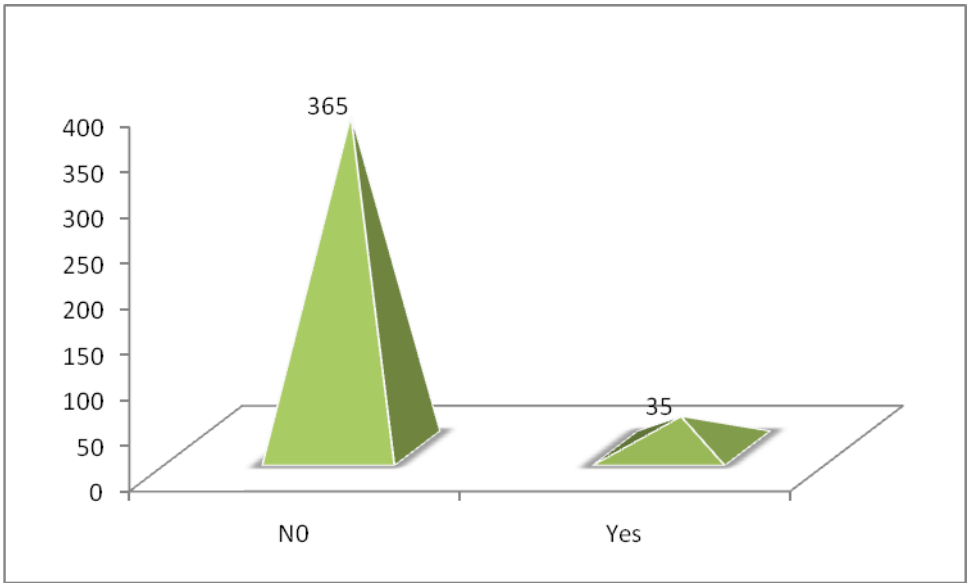
UTI	Frequency	Percent
No	356	89
Yes	44	11
Total	**400**	**100**



44 out of 400 GDM(11%) women had urinary tract infection . Out of these 44 women 32 women underwent preterm labour (72 %), giving birth to low weight babies. Most of UTI was seen in late half of 1st trimester and the 2nd trimester.

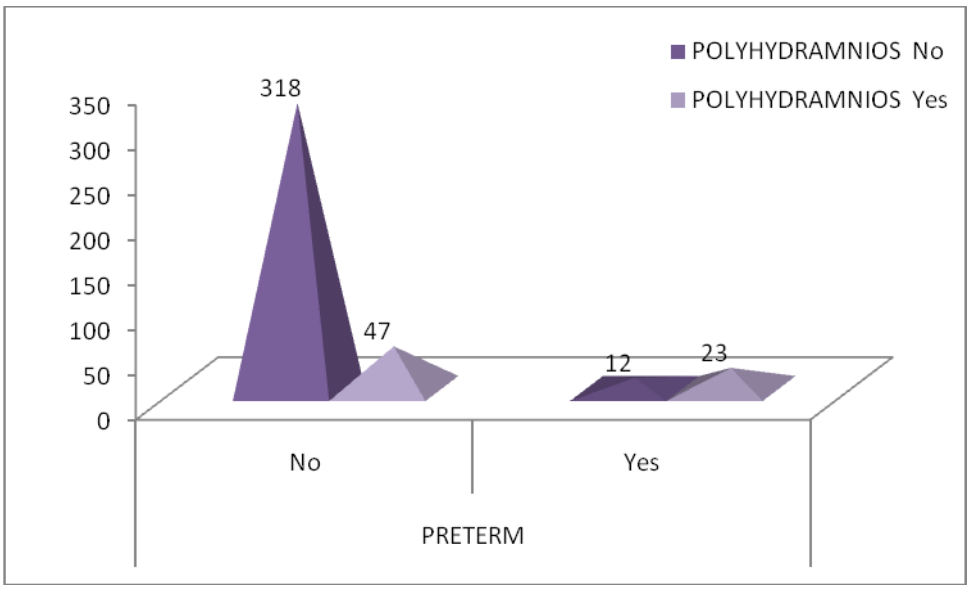
9. PRETERM LABOUR

PRETERM	Frequency	Percent
N0	365	91.2
Yes	35	8.8
Total	**400**	**100**



In this study 8.8 % had preterm labour(n= 35) . Most of them had associated complications of polyhydramnios and urinary tract infection indicating that these could be the causative factors for the preterm birth.

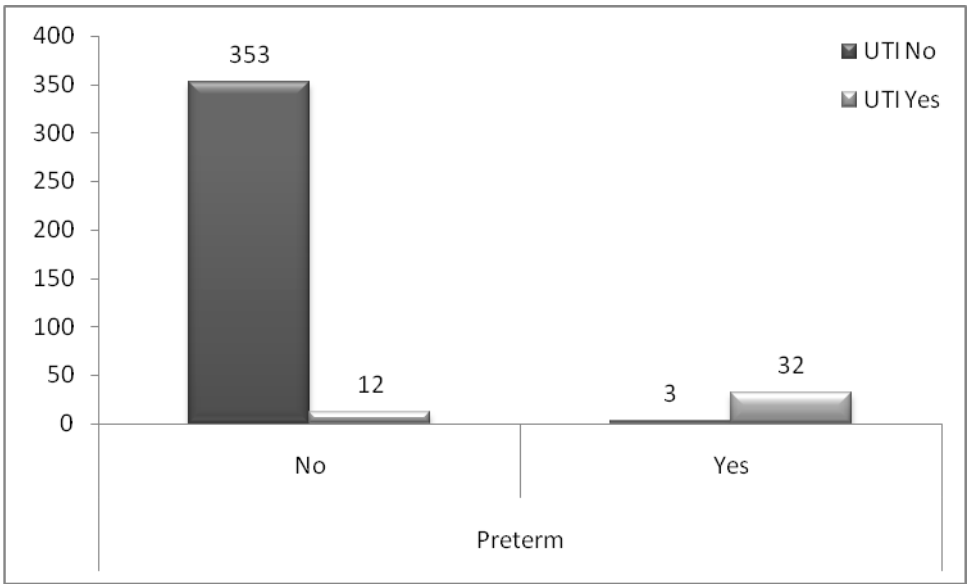
POLYHYDRAMNIOS vs PRE TERM	No	Yes	Total	Chi sq	P	OR
No	318	12	330	61.76	**0.001**	1.43
Yes	47	23	70			
Total	**365**	**35**	**400**			



Out of the 35 women who delivered preterm 23 of them had associated polyhydramnios suggesting that polyhydramnios could be the cause for preterm labour and the results were statisticaly significant (p value=0.001 chi sq=61.76) and the odds ratio was >1.

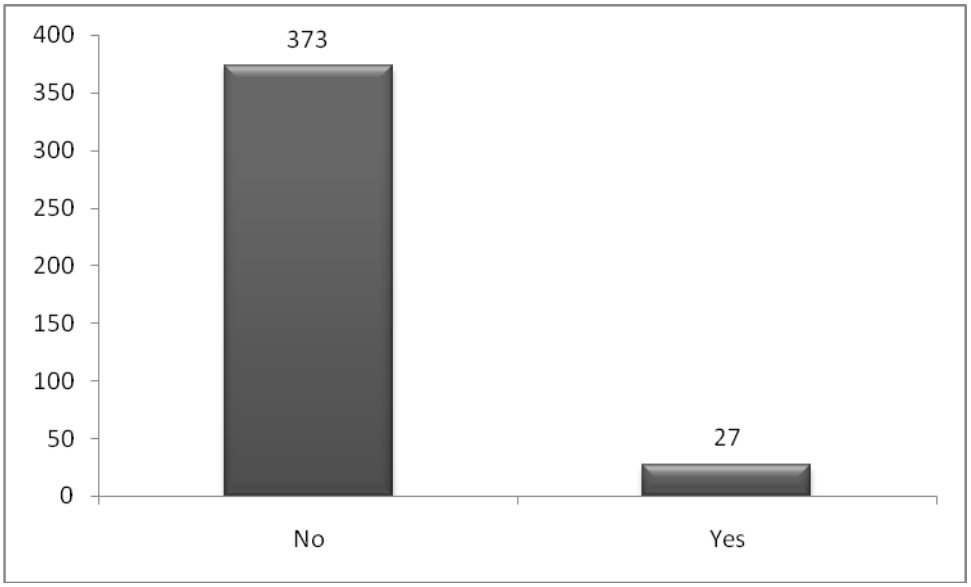
UTI vs PRE TERM	No	Yes	Total	Chi sq	P	OR
No	353	3	356	253.4	**0.001**	3.64
Yes	12	32	44			
Total	**365**	**35**	**400**			

Out of the 35 GDM women who had preterm labour,32 of them had associated UTI or previous history of UTI and the p value was statistically significant(0.001). the odds ratio was 3.64 indicating that women with UTI had three times more risk of delivering preterm than those without UTI.



10. PROM

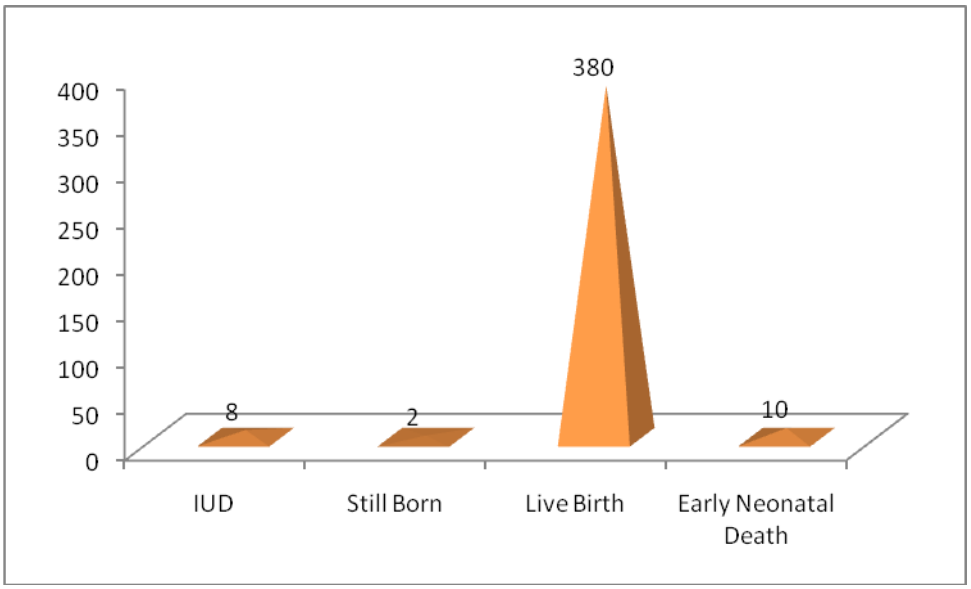
PROM	Frequency	Percent
No	373	93.2
Yes	27	6.8
Total	**400**	**100**



PROM was seen in 6.8 % of the study population . Out of 400 women 27 had premature rupture of membranes(n= 27) . In these 27 women 14 of them underwent caesarean section ad 12 delivered via labournaturalis and 1 via instrumental delivery. 2 of them had preterm premature of membranes (PPROM).Clear liquor was seen in 14 women who had PROM and the liquor was meconium stained in the remaining 13.

11. FETAL OUTCOME

Fetal Outcome	Frequency	Percent
IUD	8	2
Still Born	2	0.5
Live Birth	380	95
Early Neonatal Death	10	2.5
Total	**400**	**100**



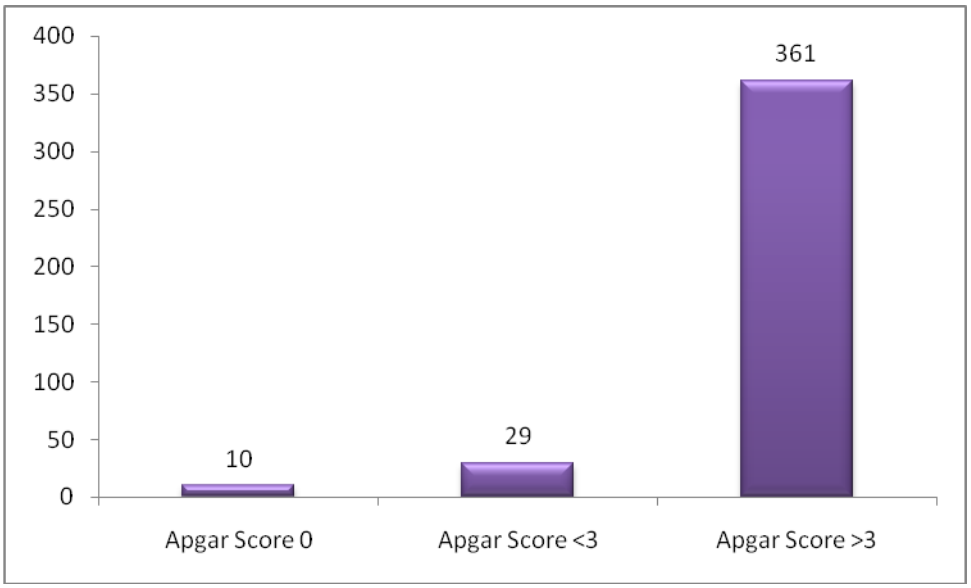
Out of the 400 GDM pregnancies , 380 were live birth (95 %) . intra uterine death was seen in 2 % (n= 8). Out of these eight, 3 of them were preterm IUDs and the remaining 5 were term IUDs.

Out of the 400 pregnanices , 2 were still born (0.5%) and early nenonatal death(within 7 days) was seen in 2.5 %. The causes of early neonatal death in

order are respirtory distress syndrome (n=5), hypoglycemia and HIE (n=4), sepsis (n=1). Out of the 10 early neonantal death, 7 had congenital anomalies and the p value was statiscally significant

12. APGAR

APGAR	Frequency	Percent
Apgar Score 0	10	2.5
Apgar Score <3	29	7.2
Apgar Score >3	361	90.2
Total	**400**	**100**



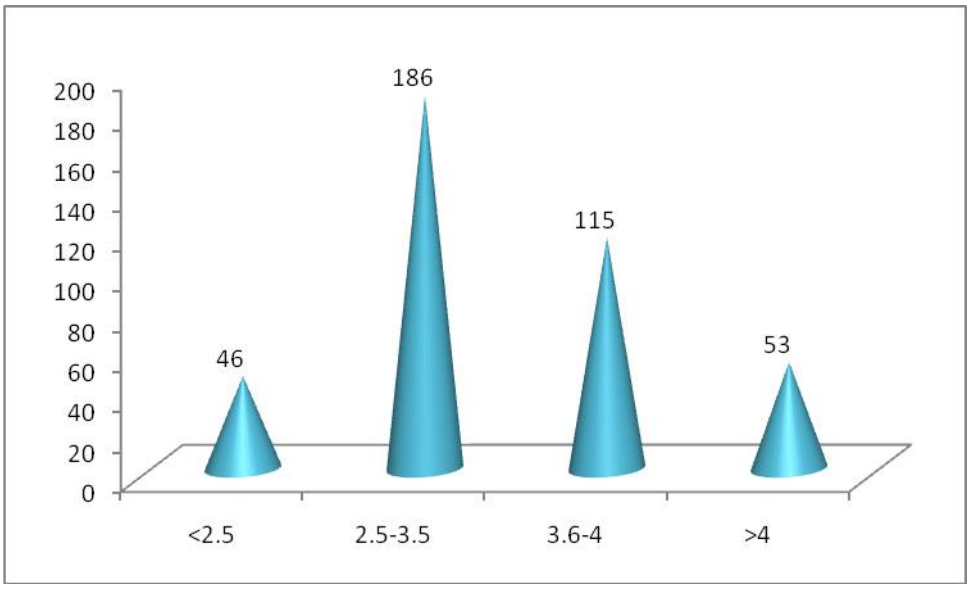
All babies with the 10 minute APGAR less than 3 were said to have birth asphyxia and 29 babies (7.2%) had birth asphyxia with the 10 minute APGAR

score less than 3. 13 out of these 29 babies had congenital anomalies . Of these 29 babies 10 expired in the early nenonatal period.

361 babies had an 10 min APGAR score > 3 (90%). APGAR score of zero was found in 10 babies which belonged to the intra uterine death and still born category.

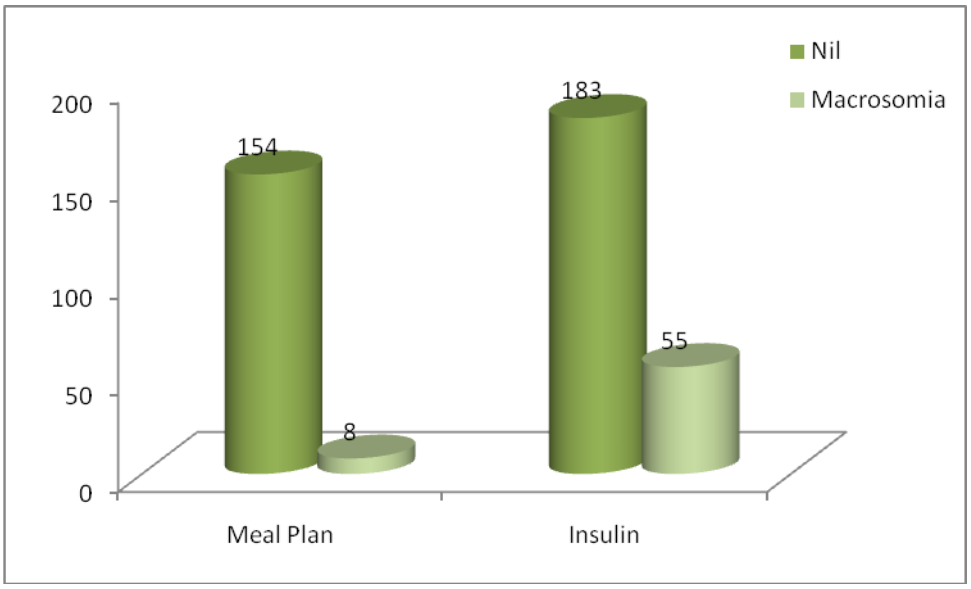
13. BIRTH WEIGHT

Birth Weight	Frequency	Percent
<2.5	46	11.6
2.5-3.5	186	46.5
3.6-4	115	28.8
>4	53	13.1
Total	**400**	**100**



Most (46.5%) of the babies born to GDM mothers had birth weight ranging from 2.5 to 3.5 kg (n =186) . 11.6 % of babies were low birth weight (n= 46) . Of these 46 babies , 35 were preterm . 28.8 % (n= 115)weighed between 3.6 to 4 kgs .

	Meal Plan	**Insulin**	**Total**	**Chi Sq**	**p**	**OR**
Nil	154	183	337	22.64	**0.001**	5.78
Macrosomia	8	55	63			
Total	162	238	400			

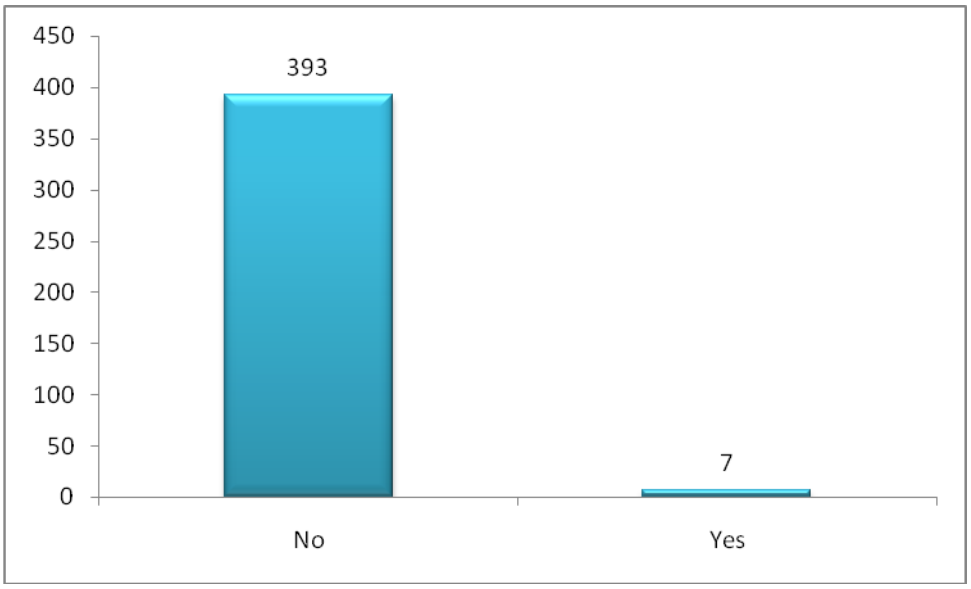


Macrosomia (> 4kg) was seen in 13.1 % of the babies (n = 55). Of these babies , 48 were born to mother who were on insulin and the p value was statistically significant (0.001).

Birth trauma and shoulder dystocia was seen in 5 babies . Out of the 55 babies who were macrosomic , 48 were delievered by LSCS, 8 via instrumental and 3 via labour naturalis.

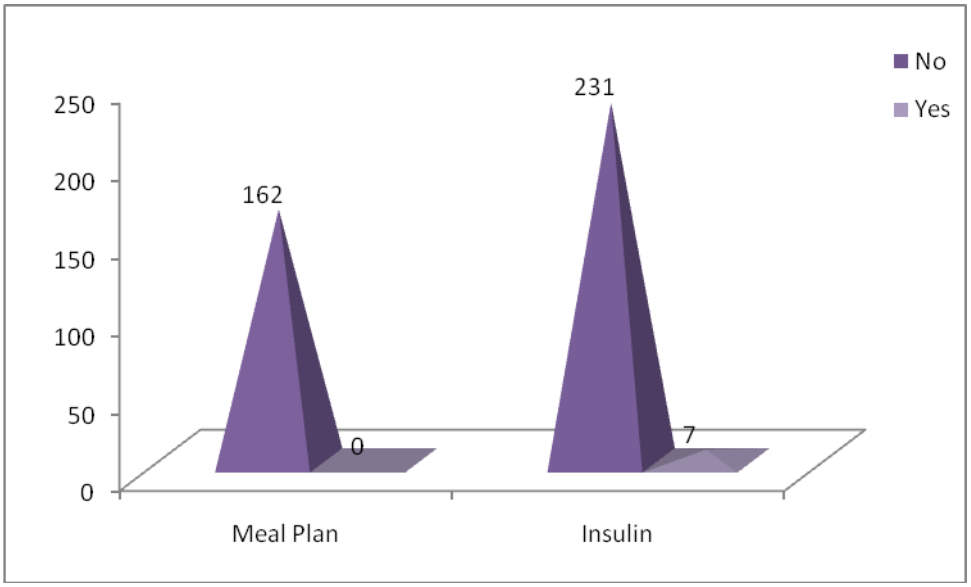
14. SHOULDER DYSTOCIA / TRAUMA

Shoulder Dystocia / Trauma	**Frequency**	**Percent**
No	393	98.2
Yes	7	1.8
Total	**400**	**100**



Out of 400 pregnancies , shoulder dystocia and birth trauma was seen in 1.8 % (n= 7). This was encountered in large babies with birth weight ranging from 3.9 to 4.3 kgs. 5 babies had birth asphyxia (10 minute APGAR < 3), however these babies improved in the neonatal period and had a good neonatal outcome.

Dystocia / Trauma	**Meal Plan**	**Insulin**	**Total**	**Chi Sq**	**P**
No	162	231	393	4.85	**0.03**
Yes	0	7	7		
Total	**162**	**238**	**400**		

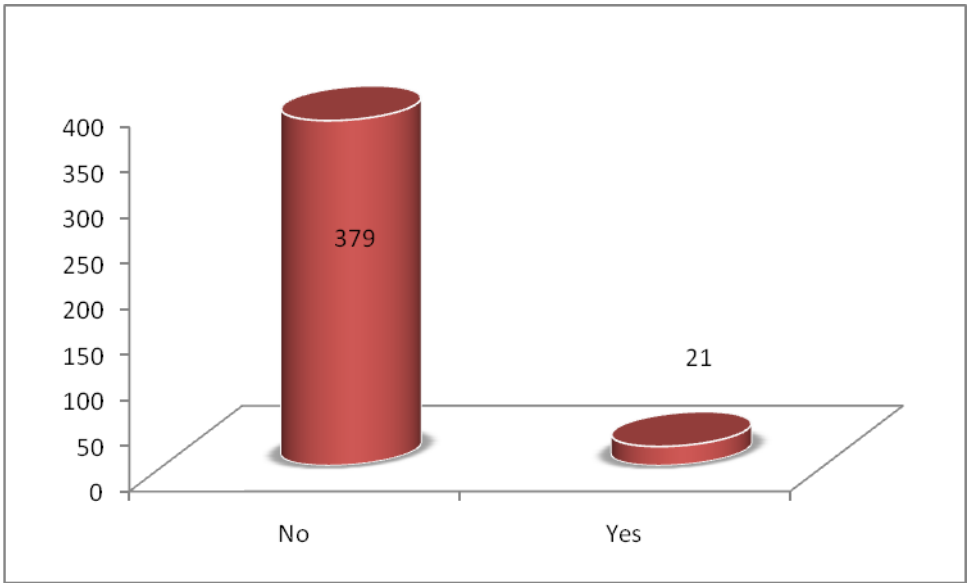


In this study it was observed that the most important cause for shoulder dystocia and birth trauma was macrosomia. Shoulder dystocia was encountered

mainly in babies ranging between 3.8kg-4.1 kg in birth weight. Out of 400 babies,7 of them had shoulder dystocia.The glycemic profile of all these 7 mothers were controlled with insulin and the p value was statistically significant(chi sq 4.85)

15. CONGENITAL ANOMALIES

CONGENITAL ANOMALIES	Frequency	Percent
No	379	94.8
Yes	21	5.2
Total	**400**	**100**

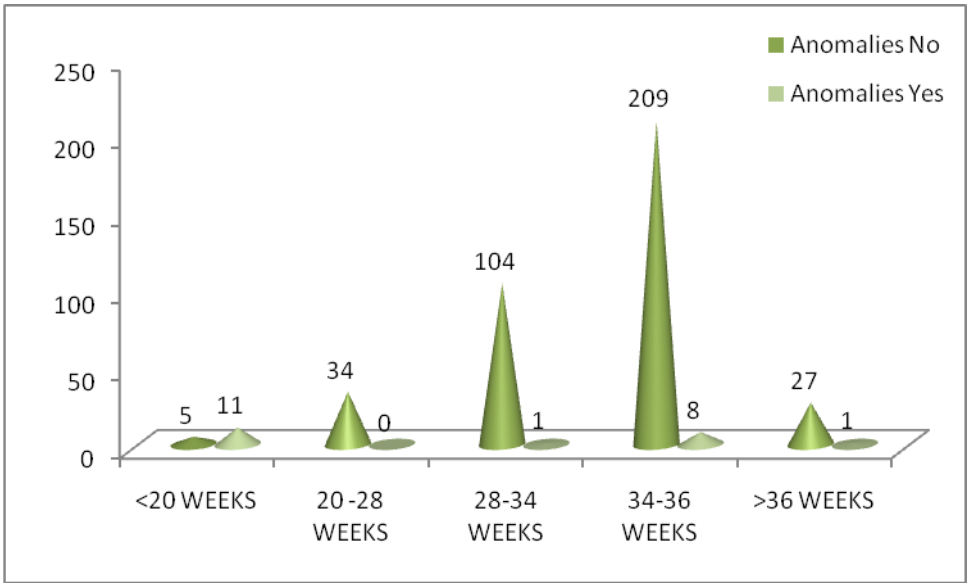


The incidence of congenital anomalies was seen in 5.2 % (21/ 400) . 50 % of these congenital anomalies occurred in early onset GDM (n =11) , where the gestation age was less than 20 weeks.

Intra uterine death was seen in 6 babies who had congenital anomalies . 7 anomalies babies suffered early neonatal death and 2 were still born . The remaining 6 babies had congenital anamoly compatible with life. The most common congenital anamoly encountered in order were

- single umbilical artery
- spina bifida,
- septal defects
- duodenal atresia

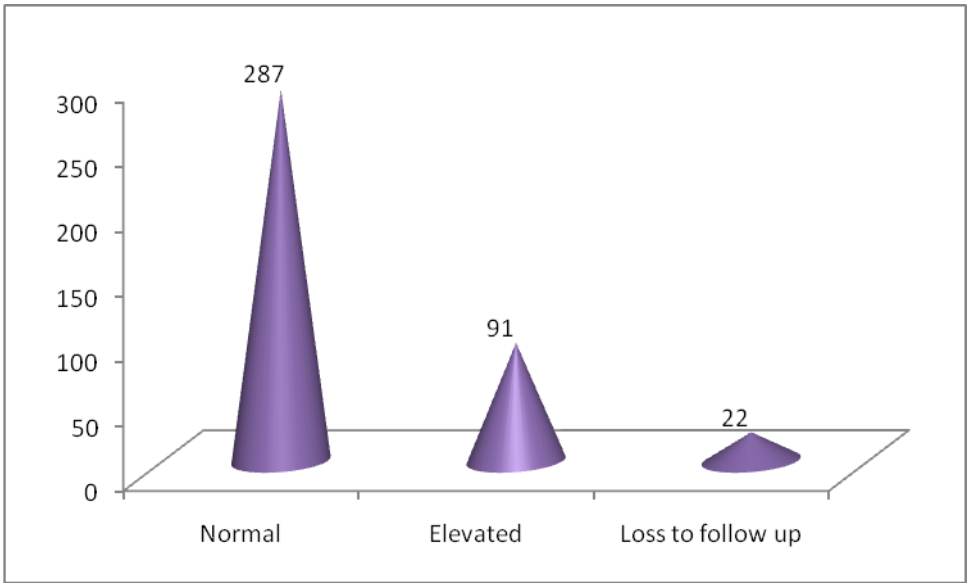
GA vs ANOMALIES	No	Yes	Total	Chi sq	P
<20 WEEKS	5	11	16	136.7	**0.001**
20 -28 WEEKS	34	0	34		
28-34 WEEKS	104	1	105		
34-36 WEEKS	209	8	217		
>36 WEEKS	27	1	28		
Total	**379**	**21**	**400**		



Congenital anomalies were present in 21 babies.Out of these 21 babies 11 babies were born to mothers with early onset GDM diagnosed before 20 weeks of gestation and the p value was statisticaly significant(p value=0.001 chi sq=136.7)

16. POSTPARTUM FOLLOW UP

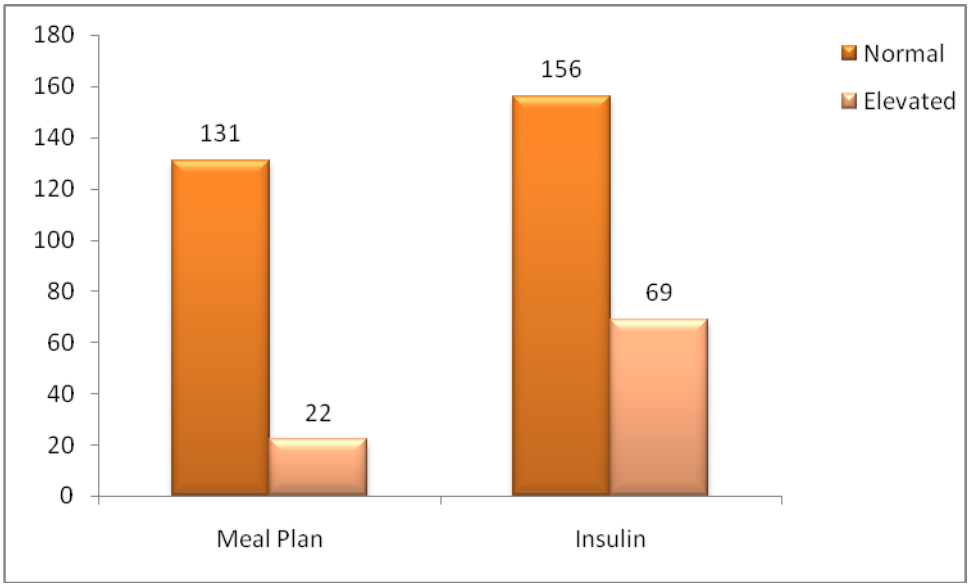
Post Partum	**Frequency**	**Percent**
Normal	287	71.8
Elevated	91	22.8
Loss to follow up	22	5.5
Total	**400**	**100**



In the post partum follow up 400 GDM mothers the attrition rate was 5 % (n=22) .Of the remaining 378 women the OGTT was performed at 6 weeks post partum and was found to be elevated in 91 women. In these 91 women, 69 women were on insulin and 22 women were on meal plan. The remaining 287 women had normal OGTT values. This implied that GDM women who were treated on insulin were glucose intolerant in the postpartum period as well and the p value was statsistically significant.

Post Partum	Meal Plan	Insulin	OR	P
Normal	131	156	2.63	**0.001**
Elevated	22	69		

The ODDS ratio was 2.63 indicating that GDM women who were treated with insulin had twice the risk of becoming glucose intolerant than those on meal plan.



DICUSSION

GDM has been diagnosed as a clinical entity for the past 50 years. Early studies have strongly indicated untreated carbohydrate intolerance during pregnancy is associated with higher rates of maternal mortality and morbidity. The purpose of screening , treatment and management of GDM is to prevent still birth, congenital anomalies, pre eclampsia , intra uterine death and decrease the incidence of macrosomic babies and cesarean section rates thereby reducing maternal and perinatal morbidity and mortality. The findings of the present study confirmed that GDM patients are liable to have adverse pregnancy outcomes.

The maximum incidence of GDM occurred between 26 to 30 years of age (32.7%). Ismail NA et al reported the maximum mean maternal age of GDM in their study was 27.9 years.

The increasing incidence was seen in higher parity which was also reported by Farook et al. In this study similar findings were observed (56.2 % in multigravida and 43.8 % in primi gravidas).

The maximum number of GDM cases were detected between 34 and 36 weeks of gestation (54.2%),which can be attributed to the fact that the maximum insulin resistance occurs at this age which was also reinforced by Peraldi et al

Mutummatou leidi[73] et al studied that caesarean section rates was higher in women with GDM (52 %). In this study, the incidence of caesarean section was higher (49 %) when compared to labournatura(46%).

Ameya R et al[72] studied the feto maternal outcomes in GDM and found that preclampsia complicating pregnancy was found in 26 % of GDM mothers. In this study also, 26 % of GDM mothers had associated GDM complicating pregnancy.

Mutummatouleidi et al observed increasing frequency of preterm labour and polyhydramnios in GDM patients. Krishnamoorthy et al studied that the incidence of pre eclampsia in GDM was 30 % and preterm labor and PROM was 9 % and 8 % respectively. In this study preterm labour was encountered in 8.8 % of the population and PROM in 6. 8%.

As far as the fetal complications were concerened congenital anomalies were encountered in 5.2% of the study population, while according to Ameya et al 8% had congenital anomalies. The incidence of macrosomia was 13.2% in this study whereas higher incidence was noted in the other studies(40% in study by Ameya et al and 23 % in study by Mutummatou et al). Adverse fetal outcome (still born,intrauterinedeath,early neonatal death)was seen in 5% of the study population and birth asphyxia in 7.2%. Shoulder dystocia was seen only in 1.8% of the study population.

During the postpartum follow up at six weeks ,22.8% of the women were glucose intolerant and those on insulin had twice the risk of being glucose intolerant than those on meal plan alone.Kjos et al[22] performed 75 gm. OGTT 5 to 8 weeks after delivery in 246 women with GDM and found 19% had abnormal OGTT, out of which 10% had impaired glucose tolerance and 9% had T2DM. 16.9% of the population were glucose intolerant in the study by Ameya et al.

CONCLUSION

To conclude,based on the observations of this study,GDM is associated with adverse complications in both the mother and fetus. A large proportion of women also progress to become overt diabetics in the future hampering with their quality of life by causing morbidity in various forms. Therefore all antenatal women attending the OPD should be offered a simple Glucose challenge test and if found negative the test has to be repeated every trimester. Once diagnosed with GDM appropriate glycemic control either via insulin or meal plan has to be achieved for good pregnancy outcome and to prevent the complications. Proper counseling should be given to the patient at the time of discharge to have her sugars checked in the postpartum period.Early detection and prompt management of this condition can tremendously reduce the short term and long term complications in both the mother and neonate.

LIMITATIONS OF THE STUDY

- The GDM mothers could be followed up only till six weeks postpartum and long term follow up was not feasible in the one year study period.
- Postpartum follow up could not be achieved in 100% of the study population as few women did not turn up for follow up.
- The cause of prior abortions or fetal loss(that could have been due to overt diabetes) remained uninvestigated in few patients and they were included in the study

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ABBREVIATIONS

GDM-Gestational Diabetes Mellitus

NIDDM-Non insulin dependant Diabetes Mellitus

IDM-Insulin dependant Diabetes Mellitus

RDS-Respiratory distress syndrome

ATP-Adenosine triphosphate

T2 DM-Type 2 Diabetes Mellitus

OGTT-Oral glucose tolerance test

GCT-Glucose challenge test

DIPSI-Diabetes in pregnancy study group India

HAPO-Hyperglycemia and adverse pregnancy outcome

IADPSG-International association of Diabetes and pregnancy study group

ACOG-American college of Obstetricians and Gynecologists

FFA-Free fatty acid

Sflt-Solubefms like tyrosine kinase

LGA-Large for gestational age

FBS-Fasting blood sugar

PPBS-Post prandial blood sugar

UTI-Urinary tract infection

PROM-Premature rupture of membranes

PROFORMA

Study No : **Hospital No. :**

Name : **Occupation :**

Age :

Address :

SOCIO- ECONOMIC STATUS:

1. CHIEF COMPLAINTS

2. HISTORY OF PRESENTING COMPLAINTS

H/o months of amenorrhea

Able to perceive fetal movements yes/no

C/o pain abdomen yes/no

C/o leaking/bleeding PV

C/o headache/blurring of vision/pedal edema

C/o oliguria / epigastric pain

c/o burning micturition yes/no

3. OBSTETRIC HISTORY

Married life

Consanguinity

Obstetric score**:** Gravida

Para

Living

Abortion

Dead

Booked/ Unbooked

Details of each pregnancy:

No of pregnancies	**Period of gestation**	**Associated complications**	**Outcome NVD/LSCS**	**Baby details**

Present pregnancy: booked at

Trimester history:

I trimester: H/O excessive vomiting/fever/UTI

Folic acid supplementation

Dating scan

II trimester: Quickening felt at

Anomaly scan

C/O headache/blurring of vision/pedal edema

C/O draining/bleeding pv

C/O burning micturiton

III trimester: Perceiving fetal movement

C/O headache/pedal edema/blurring of vision

C/O draining/bleeding pv

4. MENSTRUAL HISTORY

Age of Menarche:

Flow:

Clots:

Dysmenorrhoea:

LMP: Wks. Days

EDD: Wks. Days

5. PAST HISTORY

DIABETES/HYPERTENSION/Connective tissue disorders/Bronchial asthma/cardiac disease/thyroid disorders/blood transfusion/surgery

6. FAMILY HISTORY

TB / Bronchial Asthma / Diabetes mellitus / Hypertension / Any cancer / Bleeding disorders / Thyroid disorders

7. PERSONAL HISTORY

Diet :

Appetite :

Bowels :

Micturition :

Sleep :

8. EXAMINATION OF PATIENT:

On examination

1. Height in cms :

2. Pre pregnant Weight :

Present weight :

Weight gain :

BMI :

3. Temperature in degree

4. BP in mm of Hg

5. Respiratory rate per minute

6. Pulse rate

7. Pallor

8. Edema

9. Icterus

Systemic examination

a. CVS

b. RS

c. Per abdomen :

Inspection: abdomen-normal/over distended

Striaegravidarum :

Linea nigra :

Dilated veins/sinuses :

Palpation: Fundal height:

Abdominal Circumference :

Fundal grip :

Lateral grip :

Pelvic grip :

Liquor: adequate/scanty

Auscultation: FHS (/min)

EFW

Per vagina: cervix dilatation:

Length :

Effacement :

Position :

Consistency :

Vertex station :

Membranes :

Pelvis: adequate/not:

DIAGNOSIS:

Investigations

Hb

RBS

PCV

Blood group

Urine routine: albumin/ Sugar

BT

CT

HIV

HBsAg

VDRL

Glucose challenge test

FBS/PPBS

Blood urea

Serum Creatinine

Serum Uric acid

LFT

Platelet count:

USG

Fetal ECHO

Electrocardiogram

Fundoscopy

Treatment: Insulin/Meal Plan

Pregnancy outcome:

- Gestational age at delivery
- Vaginal delivery: assisted/ spontaneous
- Caesarean section
- Maternal complications

Perinatal outcome:

- Live/Still birth/IUD
- Congenital anomalies
- 10 min APGAR
- Birth weight
- Birth trauma
- Neonatal Deaths

POSTPARTUM OGTT- Elevated/Not elevated

INFORMATION SHEET

Place of Study **:** Chengalpattu Medical College Hospital

Name of the Investigator: Dr. Nithya.V

Name of the Participant : **Age:** **Hospital No:**

We are conducting a study on **"FETOMATERNAL OUTCOME OF GESTATIONAL DIABETES MELLITUS"** The purpose of the study is to identify the antepartum intrapartum and postpartum complications and their incidence in patients with gestational diabetes mellitus and to study the outcome of pregnancy in these patients and the incidence of GDM patients turning to overt diabetics during the postpartum follow up after six weeks.

The privacy of the patient in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research , no personally identifiable information will be shared.

Taking part in the study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.
- You are invited to take part in this study. The information in the document is meant to help you decide whether or not to take part . Please feel free to ask if you have any queries or concerns.
- We have obtained approval from the institutional ethical committee

PRINCIPAL INVESTIGATOR

DR.NITHYA.V

II year MS post graduate,

Department of Obstetrics and Gynaecology

Chengalpattu medical college, Chengalpattu.

Signature of investigator **Signature of patient /guardian**

Date :

Chengalpattu

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செங்கல்பட்டு அரசு பொது மருத்துவமனையில் மகப்பேறு மற்றும் மகளிர் நல துறையில் ஆராய்ச்சி நடைபெற்றுவருகின்றது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்பிகிறோம். இதனால் உங்களுக்கு எந்த பாதிப்பும் ஏற்படாது என்பதை தெரிவித்துக் கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதுஅல்லது ஆராய்ச்சியின் போது உங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில் தான் இருக்கிறது. மேலும்நீங்கள் எந்தநேரமும்இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

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MASTER CHART

MATERNAL COMPLICATIONS												FETAL COMPLICATIONS					
S.NO	NAME	AGE	PARITY	GEST AGE AT DIAGNOSIS <20 weeks-1 20-28 weeks-2 28-34 weeks-3 34-36 weeks-4 >36 weeks-5	TREATMENT	POLYHYDRAMNIOS	PRE ECLAMPSIA ABSENT-1 MILD PRE ECLAMPSIA-2 SEVERE PRE ECLAMPSIA-3	UTI	PRETERM LABOUR	PROM	MODE OF DELIVERY	FETAL OUTCOME IUD-1 STILL BORN-2 LIVE BIRTH-3 EARLY NEONATAL DEATH-4	BIRTH WEIGHT	APGAR SCORE 0-1 10 MIN APGAR<3-2 10MIN APGAR>3-3	SHOULDER DYSTOCIA AND BIRTH TRAUMA	ANOMALIES	POSTPARTUM 6 WEEKS OGTT NORMAL-1 ELEVATED-2 LOSS TO FOLLOW UP-3
1	JEMIMA	18	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	INSTRUMENTAL	3	3	3	NO	NO	1
2	ARULMARY	23	G2A1	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
3	RADHA	31	G3P2L2	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	2.75	3	NO	NO	1
4	PRIYA	25	PRIMI	1	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	4	2	NO	YES	3
5	KAMATCHI	19	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	1
6	SARGUNAM	23	PRIMI	3	INSULIN	YES	1	YES	YES	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
7	KANCHANA	28	G2P1L1	3	MEAL PLAN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	2.6	3	NO	NO	1
8	RASIKA	36	G3P2L2	2	INSULIN	YES	2	YES	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	2
9	PREETHI	23	PRIMI	1	INSULIN	NO	3	NO	NO	NO	ELECTIVE LSCS	4	4.2	2	NO	YES	1
10	VIMALA	25	G3A2	5	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	1
11	ANITHA	20	PRIMI	4	MEAL PLAN	NO	2	NO	NO	NO	INSTRUMENTAL	3	3	3	NO	NO	1
12	KAVITHA	26	G2P1L1	3	INSULIN	NO	2	NO	NO	NO	LABOUR NATURA	3	2.75	3	NO	NO	1
13	MEENA	26	G2P1L1	3	MEAL PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.2	3	NO	NO	1
14	LATHA	18	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	1	3.6	1	NO	NO	1
15	PRIYA	30	G3P1L1A1	2	INSULIN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	2.9	3	NO	NO	2
16	YEMHA	31	G2P1L1	2	MEAL PLAN	YES	1	YES	YES	NO	LABOUR NATURA	3	2.4	3	NO	NO	2
17	VASANTHA	36	G3P2L2	5	INSULIN	YES	1	NO	NO	NO	LABOUR NATURA	3	2.75	2	NO	NO	3

18	KAVIYA	23	PRIMI	2	MEAL PLAN	YES	2	YES	YES	NO	LABOUR NATURA	3	2.2	3	NO	NO	1
19	SAISKALA	25	G2A1	1	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	1	3	1	NO	YES	1
20	RAMYA	18	PRIMI	2	INSULIN	YES	2	YES	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	1
21	JILLY	35	G3P2L2	2	INSULIN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	2.6	3	NO	NO	2
22	POONAM	26	G2P1L1	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	1
23	SWETHA	32	PRIMI	4	INSULIN	YES	1	NO	NO	NO	EMERGENCY LSCS	3	4.3	3	NO	NO	1
24	VARSHA	28	G2P1L1	3	MEAL PLAN	YES	1	NO	YES	NO	LABOUR NATURA	3	2.3	2	NO	NO	2
25	QUEEN	30	G2P1L1	2	INSULIN	NO	1	NO	NO	YES	LABOUR NATURA	3	3.6	3	NO	NO	2
26	BINA	33	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.75	3	NO	NO	2
27	UMA	26	G2P1L1	5	INSULIN	NO	2	NO	NO	NO	ELECTIVE LSCS	3	4.5	2	NO	NO	1
28	SANKARI	18	PRIMI	3	MEAL PLAN	YES	1	YES	YES	NO	LABOUR NATURA	1	2.3	1	NO	NO	1
29	MEENA	24	G3A2	1	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	2.6	3	NO	NO	1
30	SARANYA	28	G3P2L2	3	MEAL PLAN	YES	2	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	1
31	SHANTHI	31	PRIMI	2	INSULIN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.9	3	NO	NO	2
32	SARALA	23	PRIMI	2	MEAL PLAN	YES	1	YES	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	1
33	NIRMALA	33	G2P1L1	2	INSULIN	YES	1	NO	NO	NO	ELECTIVE LSCS	3	2.7	3	NO	NO	2
34	SANTHIYA	19	PRIMI	4	MEAL PLAN	NO	1	NO	NO	YES	LABOUR NATURA	3	2.4	3	NO	NO	1
35	REVATHI	28	PRIMI	2	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.9	3	NO	NO	1
36	RATHIGA	25	G2P1L1	3	MEAL PLAN	YES	1	NO	NO	NO	EMERGENCY LSCS	3	3	3	NO	NO	1
37	YAMUNA	31	PRIMI	5	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.75	3	NO	NO	1
38	SUNGAVI	26	G2P1L1	2	INSULIN	YES	2	NO	NO	NO	ELECTIVE LSCS	3	3	3	NO	NO	1
39	SHANTHI PRIYA	36	G3P2L2	2	INSULIN	NO	1	YES	YES	NO	LABOUR NATURA	3	2.2	3	NO	NO	2
40	THENMOZHI	30	G2P1L1	3	MEAL PLAN	NO	1	YES	YES	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
41	VASANTHI	35	G3P2L2	2	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	1
42	DEVI	20	PRIMI	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.6	3	NO	NO	1
43	KANCHANA	26	G2P1L1	3	MEAL PLAN	YES	1	NO	NO	NO	EMERGENCY LSCS	3	2.75	3	NO	NO	1
44	KALAIYARASI	24	PRIMI	2	INSULIN	YES	1	NO	NO	NO	EMERGENCY LSCS	3	2.8	3	NO	NO	1
45	MOHANOPRIYA	37	G2P1L1	4	INSULIN	YES	1	NO	NO	NO	EMERGENCY LSCS	3	3.5	3	NO	NO	2
46	SURYA DEVI	30	G2P1L1	3	MEAL PLAN	NO	1	YES	YES	NO	LABOUR NATURA	3	2.2	3	NO	NO	1
47	SARANYA	23	PRIMI	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
48	KALAIVANI	19	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.7	3	NO	NO	1
49	MEENA	26	G2P1L1	4	INSULIN	YES	3	NO	NO	NO	LABOUR NATURA	3	2.75	3	NO	NO	2
50	LAKSHMI	33	G3P2L2	5	INSULIN	YES	1	NO	NO	NO	ELECTIVE LSCS	3	2.6	3	NO	NO	1
51	SARALA	28	G3P1L1A1	3	MEAL PLAN	NO	1	NO	NO	NO	INSTRUMENTAL	3	4	3	NO	NO	2
52	SURYA KANDHI	25	PRIMI	2	INSULIN	YES	3	YES	YES	YES	LABOUR NATURA	3	2.2	3	NO	NO	1
53	LAILA	18	G2A1	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.9	3	NO	NO	1
54	VANITHA	24	PRIMI	1	MEAL PLAN	YES	1	NO	NO	NO	EMERGENCY LSCS	2	2.9	1	NO	YES	1
55	SURYA KALA	33	G2P1L1	4	INSULIN	YES	2	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	2
56	MANO PRIYA	30	G2P1L1	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
57	SIVASHINI	28	PRIMI	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	3
58	SAINDEVI	36	G3P1L1A1	3	INSULIN	YES	3	NO	YES	NO	LABOUR NATURA	3	2.4	3	NO	NO	2

59	LAILA	26	PRIMI	3	MEAL PLAN	YES	1	NO	NO	NO	LABOUR NATURA	3	2.7	3	NO	NO	1
60	REVATHI SREE	35	G2A1	5	INSULIN	YES	3	NO	NO	YES	EMERGENCY LSCS	3	3.7	3	NO	NO	2
61	GOMATHI	31	G2P1L1	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.4	2	NO	NO	1
62	SEELA	30	G3P2L2	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	4	2.8	2	NO	NO	2
63	LAKSHMI	20	PRIMI	3	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.8	3	NO	NO	1
64	KANAGAVATHI	26	G2P1L1	2	INSULIN	YES	1	NO	NO	NO	ELECTIVE LSCS	3	2.5	3	NO	NO	2
65	SURYAGANDHI	32	G2A1	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
66	ARASI	26	G2P1L1	3	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.5	3	NO	NO	1
67	MAYIL	33	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	2.6	3	NO	NO	1
68	PRITIKA	23	PRIMI	1	INSULIN	YES	1	NO	NO	YES	LABOUR NATURA	3	2.4	2	NO	YES	1
69	YAMINI	37	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	4	3.8	2	NO	NO	1
70	SUDHA	26	G3P1L1A1	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.3	3	NO	NO	2
71	CHITHRA	24	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.9	3	NO	NO	1
72	GAJAVALLI	26	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3	3	NO	NO	1
73	MALAR	18	PRIMI	1	INSULIN	YES	3	YES	YES	NO	LABOUR NATURA	2	2.4	1	NO	YES	1
74	AMMU	25	PRIMI	4	MEAL PLAN	NO	1	YES	NO	YES	EMERGENCY LSCS	3	3	3	NO	NO	1
75	NIVETHA	33	G3P2L2	3	INSULIN	YES	1	NO	NO	NO	LABOUR NATURA	3	2.3	3	NO	NO	2
76	PERIYANAYAKI	26	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.9	3	NO	NO	1
77	THENAMBAL	28	PRIMI	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.3	3	NO	NO	2
78	KANAMA	35	G3A2	2	INSULIN	YES	3	NO	NO	NO	ELECTIVE LSCS	3	2.6	3	NO	NO	2
79	DILLIRANI	36	G2P1L1	5	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.5	3	NO	NO	1
80	MANONMANI	24	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.6	2	NO	NO	1
81	SRIPRIYA	19	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.5	3	NO	NO	1
82	LAVANYA	27	G3P2L2	3	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.1	3	NO	NO	3
83	KAVITHA	31	G2P1L1	3	INSULIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	2.9	3	NO	NO	1
84	MALINI	26	PRIMI	4	INSULIN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.8	3	NO	NO	1
85	MANJULA	24	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	2.6	3	NO	NO	1
86	SAMITHRA	33	G3P2L2	4	INSULIN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	3	3	NO	NO	2
87	KANAGA	23	G2P1L1	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
88	KARPAGAM	28	PRIMI	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.2	2	NO	NO	1
89	THAMARAI	18	PRIMI	3	MEAL PLAN	NO	1	NO	NO	YES	EMERGENCY LSCS	3	3	3	NO	NO	1
90	NANTHINI	25	PRIMI	5	INSULIN	YES	1	NO	NO	NO	LABOUR NATURA	3	2.4	2	NO	NO	1
91	VAIDEGI	32	G3P2L2	2	INSULIN	YES	3	YES	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	2
92	MUNIYAMMAL	22	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	1
93	KASIYAMMAL	21	PRIMI	1	MEAL PLAN	YES	1	YES	YES	NO	LABOUR NATURA	1	2.2	1	NO	YES	1
94	SHOBANA	20	PRIMI	3	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.2	3	NO	NO	3
95	ANISHIYA	24	G2P1L1	3	MEAL PLAN	YES	1	NO	NO	YES	EMERGENCY LSCS	3	3	3	NO	NO	2
96	SRI DEVI	26	PRIMI	4	INSULIN	NO	3	NO	NO	NO	INSTRUMENTAL	3	4	2	YES	NO	2
97	MAYA	35	G2P1L1	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.6	3	NO	NO	1
98	SUSILI	31	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	2.75	3	NO	NO	1

99	DEVI KALA	24	PRIMI	3	INSULIN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	2
100	SWATHI	28	G2P1L1	1	MEAL PLAN	YES	1	YES	YES	NO	LABOUR NATURA	1	2.3	1	NO	YES	1
101	AMALA	23	PRIMI	5	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.8	3	NO	NO	1
102	SUILA	19	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	1
103	MEENA	27	G2P1L1	3	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	4.2	3	NO	NO	2
104	CHITHRA	33	G3P2L2	4	INSULIN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.9	3	NO	NO	2
105	AMBIGA AMMAL	26	PRIMI	3	MEAL PLAN	YES	1	NO	NO	NO	LABOUR NATURA	3	2.6	3	NO	NO	1
106	PRIYA SREE	36	G2P1L1	1	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	4	3.2	2	NO	YES	2
107	SREEWATHI	28	G2P1L1	3	INSULIN	NO	1	NO	NO	YES	EMERGENCY LSCS	3	4.3	3	NO	NO	1
108	SANGEETHA	24	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.8	3	NO	NO	1
109	DANALAKSHMI	18	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	4	3	2	NO	NO	1
110	KOKILA	22	PRIMI	4	INSULIN	YES	3	NO	NO	NO	EMERGENCY LSCS	3	2.9	3	NO	NO	1
111	PURNIMA	25	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.75	3	NO	NO	1
112	RAGINI	32	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	2
113	SULOCHANA	26	G2P1L1	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.6	3	NO	NO	2
114	SANGEETHA	35	G3P2L2	4	INSULIN	YES	1	NO	NO	NO	ELECTIVE LSCS	3	3.8	3	NO	NO	1
115	BOOMIKA	24	PRIMI	5	INSULIN	NO	3	NO	NO	NO	INSTRUMENTAL	3	4.1	2	YES	NO	1
116	KILIYAMA	31	G2P1L1	3	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.1	3	NO	NO	2
117	POOJA	23	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	4	3	NO	NO	1
118	BARANI	30	G2P1L1	3	INSULIN	NO	1	NO	NO	YES	EMERGENCY LSCS	3	2.9	3	NO	NO	1
119	AMMAN	33	G2P1L1	1	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	2
120	PACHAYAMAL	30	G2P1L1	3	INSULIN	YES	1	NO	NO	NO	EMERGENCY LSCS	3	2.8	3	NO	NO	1
121	GOBINDAMAL	36	G2P1L1	4	INSULIN	NO	2	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	2
122	LAXMISRI	26	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3	3	NO	NO	1
123	JANANI	28	PRIMI	3	MEAL PLAN	NO	1	YES	YES	NO	LABOUR NATURA	3	2.2	3	NO	NO	2
124	ANUPAMA	24	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
125	MERCY	30	G3P2L2	3	INSULIN	YES	3	NO	NO	NO	ELECTIVE LSCS	3	2.6	3	NO	NO	1
126	USHA	20	PRIMI	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.9	3	NO	NO	1
127	AMMU	30	G2P1L1	3	MEAL PLAN	NO	2	NO	NO	NO	LABOUR NATURA	3	2.7	3	NO	NO	2
128	SATHIYA PRIYA	32	G2P1L1	3	INSULIN	YES	1	YES	YES	NO	LABOUR NATURA	3	2.2	3	NO	NO	1
129	SATHIYA	30	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3	3	NO	NO	1
130	MERSHI	19	PRIMI	3	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.75	3	NO	NO	1
131	RUBHA	26	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	YES	EMERGENCY LSCS	3	3.4	3	NO	NO	2
132	KANNAMMAL	33	G2P1L1	1	INSULIN	YES	3	NO	NO	NO	LABOUR NATURA	3	2.8	3	NO	NO	2
133	FARTHIMA	24	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	INSTRUMENTAL	3	4.3	3	NO	NO	1
134	YAMUNA DEVI	18	PRIMI	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.2	3	NO	NO	1
135	SEELA	25	G2P1L1	4	MEAL PLAN	NO	2	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
136	KANMANI	40	G2P1L1	1	INSULIN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	2.8	2	NO	YES	2
137	VIJAYA SHANTHI	30	G2P1L1	2	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	2
138	DIVYA	31	G2P1L1	3	MEAL PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	2
139	RANCHANI	24	PRIMI	4	MEAL PLAN	YES	1	NO	NO	NO	LABOUR NATURA	3	3.1	3	NO	NO	3

140	SASIKALA	35	G2P1 L1	4	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.2	3	NO	NO	1
141	MUGAMPI GAI	23	G2P1 L1	3	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	1
142	BHAGIYALA KSHMI	20	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	2.9	3	NO	NO	1
143	ANANTHI	30	G3P2 L2	3	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.8	3	NO	NO	1
144	AMBIGA DEVI	28	PRIM I	4	MEA L PLAN	YES	1	NO	NO	YES	EMERGENC Y LSCS	3	3.2	3	NO	NO	2
145	ANU PRIYA	33	G2P1 L1	3	INSU LIN	NO	1	NO	NO	NO	INSTRUME NTAL	3	4.2	3	NO	NO	1
146	ANUTHA	26	PRIM I	3	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	3
147	RANI	37	G2P1 L1	5	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.3	3	NO	NO	2
148	SUMITHRA	30	G2P1 L1	4	MEA L PLAN	NO	2	NO	NO	YES	EMERGENC Y LSCS	3	2.6	3	NO	NO	1
149	DEVI	24	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
150	SWATHA SREE	30	G2P1 L1	4	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	4.1	2	YES	NO	1
151	KALAIVANI	31	G2P1 L1	3	MEA L PLAN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	2.75	3	NO	NO	1
152	MANJULA	30	G2P1 L1	1	MEA L PLAN	YES	1	NO	NO	NO	INSTRUME NTAL	4	2.8	2	NO	YE S	1
153	MUNIYAM MAL	35	G2P1 L1	4	INSU LIN	NO	1	YE S	NO	NO	LABOUR NATURA	3	3	3	NO	NO	2
154	KANNAGI	18	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	2.7	3	NO	NO	1
155	GOGULA PIRYA	30	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	2
156	SANKARI	23	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	2.8	3	NO	NO	1
157	SANMATHI	32	PRIM I	3	MEA L PLAN	YES	1	YE S	YES	NO	LABOUR NATURA	3	2	3	NO	NO	1
158	RUDHRA	24	G2P1 L1	1	INSU LIN	NO	1	NO	NO	YES	EMERGENC Y LSCS	3	3.6	3	NO	NO	1
159	THENMOZH I	28	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.9	3	NO	NO	1
160	YAMUNA	31	G2P1 L1	5	INSU LIN	YES	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	2
161	MALATHI	26	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.2	3	NO	NO	2
162	RAJAKUMA RI	25	G2P1 L1	4	MEA L PLAN	NO	2	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	1
163	MAHALASH MI	20	G2P1 L1	4	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	4	3.4	2	NO	YE S	3
164	KAMATCHI	30	G3P2 L2	4	MEA L PLAN	YES	1	NO	NO	NO	EMERGENC Y LSCS	3	3.2	3	NO	NO	2
165	KAYALVIZHI	33	G2P1 L1	4	INSU LIN	NO	2	NO	NO	NO	ELECTIVE LSCS	3	2.5	3	NO	NO	2
166	OVIYA	19	PRIM I	1	MEA L PLAN	NO	1	YE S	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	1
167	NEELAMBA RI	30	G2P1 L1	4	MEA L PLAN	YES	1	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1
168	SONAM	24	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	4.3	3	NO	NO	1
169	RAHINI	30	G2P1 L1	4	INSU LIN	NO	1	NO	NO	YES	LABOUR NATURA	3	3.4	3	NO	NO	2
170	KANNIYAM AL	20	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.9	3	NO	NO	1
171	KRISHNAVE NI	26	PRIM I	4	MEA L PLAN	NO	3	NO	NO	NO	EMERGENC Y LSCS	3	3.2	3	NO	NO	1
172	ARTHI	28	G2P1 L1	3	INSU LIN	YES	1	YE S	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	2
173	DEEPA	31	G3P2 L2	2	INSU LIN	NO	2	NO	NO	NO	ELECTIVE LSCS	3	2.75	3	NO	NO	2
174	JAYA	28	PRIM I	4	INSU LIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	3	3	NO	NO	1
175	TARA	30	G2P1 L1	4	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.2	3	NO	NO	1
176	DHANUSHG A	20	PRIM I	3	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	1
177	BHARATHI	24	G2P1 L1	4	INSU LIN	NO	1	YE S	NO	NO	EMERGENC Y LSCS	3	4.2	3	NO	NO	2
178	KAVERI	33	G2P1 L1	4	INSU LIN	NO	3	NO	NO	NO	EMERGENC Y LSCS	3	3.2	3	NO	NO	2
179	KANIMOZHI	32	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.1	3	NO	NO	2

180	DIVA DHARSANI	25	G2P1 L1	4	INSU LIN	NO	2	NO	NO	NO	EMERGENC Y LSCS	3	3.4	3	NO	NO	2
181	GEETHA	23	G2P1 L1	3	MEA L PLAN	NO	1	YE S	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	1
182	SELAM	28	G3P2 L2	2	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.3	3	NO	NO	1
183	VANI	20	PRIM I	5	INSU LIN	YES	2	NO	NO	YES	LABOUR NATURA	3	3.2	3	NO	NO	1
184	GOWTHAM I	31	G2P1 L1	4	MEA L PLAN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	1
185	KUMARI	22	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	INSTRUME NTAL	3	4.1	3	NO	NO	2
186	PORKODI	33	G2P1 L1	2	MEA L PLAN	YES	1	YE S	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	1
187	KAMACHI	26	PRIM I	2	INSU LIN	YES	3	NO	NO	NO	ELECTIVE LSCS	3	4.2	3	NO	NO	2
188	KEERTHIKA	37	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	3.2	3	NO	NO	1
189	KARTHIGA	35	G3P2 L2	3	INSU LIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	2.75	3	NO	NO	1
190	KURUPA	24	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	3
191	LAVANYA	39	G3P2 L2	3	INSU LIN	NO	1	N0	NO	NO	EMERGENC Y LSCS	3	4.2	3	NO	NO	2
192	KODIYTHA	21	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
193	VALRMATH I	26	G2P1 L1	4	INSU LIN	NO	3	NO	NO	NO	EMERGENC Y LSCS	3	3.2	3	NO	NO	2
194	NATHIYA	20	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.9	3	NO	NO	1
195	SUGANYA	28	G2P1 L1	4	INSU LIN	YES	3	NO	NO	NO	ELECTIVE LSCS	3	2.9	3	NO	NO	2
196	SAMBAVI	27	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.9	3	NO	NO	2
197	MANIMEGA LAI	31	G2P1 L1	5	INSU LIN	NO	2	N0	NO	NO	ELECTIVE LSCS	3	2.8	3	NO	NO	1
198	KALIYAMM AL	21	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	1
199	BHUVANA	32	G2P1 L1	3	MEA L PLAN	NO	3	YE S	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	3
200	SANTHANA M	27	G2P1 L1	4	INSU LIN	YES	1	NO	NO	NO	EMERGENC Y LSCS	3	3.6	3	NO	NO	2
201	PUVIYARAS HI	20	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
202	PANCHALAI	24	G2P1 L1	3	INSU LIN	NO	3	NO	NO	NO	EMERGENC Y LSCS	3	2.75	3	NO	NO	2
203	TAMILISAI	23	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.7	3	NO	NO	1
204	THULASI	33	G3P2 L2	4	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.4	3	NO	NO	1
205	BHUNESHW ARI	25	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	INSTRUME NTAL	3	3	3	NO	NO	2
206	SUMITHA	28	G3P2 L2	2	INSU LIN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	2
207	SARANYA	31	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	2.9	3	NO	NO	2
208	DEVI	28	G3P2 L2	4	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.3	3	NO	NO	1
209	PRIYANGA	30	G2P1 L1	3	INSU LIN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	2
210	MUNIYAM MAL	20	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	2.7	3	NO	NO	1
211	NILA	26	PRIM I	4	INSU LIN	NO	1	NO	NO	YES	LABOUR NATURA	3	3.9	3	NO	NO	1
212	SINDEVI	34	G2P1 L1	5	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	2.75	3	NO	NO	2
213	DIYA	28	PRIM I	4	INSU LIN	NO	3	NO	NO	NO	LABOUR NATURA	3	2.8	3	NO	NO	2
214	NATHIYA	19	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	3.7	3	NO	NO	1
215	SARANYA	28	G2P1 L1	3	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.1	3	NO	NO	1
216	KALAVATHI	24	G2P1 L1	4	INSU LIN	NO	1	YE S	NO	NO	ELECTIVE LSCS	3	3.4	3	NO	NO	1
217	SARAN GEETHA	20	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.7	3	NO	NO	3
218	DEVIGA SREE	25	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	4.3	3	NO	NO	1
219	LEETHA WATHI	30	G2P1 L1	3	INSU LIN	YES	3	NO	NO	YES	EMERGENC Y LSCS	3	2.6	3	NO	NO	2
220	JOHNSHI	32	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	1

221	SHANIYA	30	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.8	3	NO	NO	1
222	SANKARI	35	G2P1 L1	4	INSU LIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	4.1	3	NO	NO	1
223	MEENA	26	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	INSTRUME NTAL	3	2.7	3	NO	NO	1
224	SIVA SHANKARI	20	PRIM I	2	INSU LIN	YES	2	YE S	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	1
225	RAMANI	28	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	2.7	3	NO	NO	1
226	JAYA SUDHA	31	G2P1 L1	4	INSU LIN	NO	2	NO	NO	NO	ELECTIVE LSCS	3	4.3	3	NO	NO	1
227	THILAGAVA THI	24	PRIM I	3	INSU LIN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	3	3	NO	NO	1
228	MUNIYAM MAL	39	G2P1 L1	4	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.4	3	NO	NO	2
229	GOVINDHA MMAL	30	G3P2 L2	4	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.2	3	NO	NO	1
230	RENUGA	24	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	2.9	3	NO	NO	1
231	ILLAVATHI	20	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
232	RANGANAY AGI	24	PRIM I	4	INSU LIN	YES	3	NO	NO	YES	EMERGENC Y LSCS	3	3.2	3	NO	NO	1
233	YASODHA	23	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	1
234	GANGA	34	G2P1 L1	4	INSU LIN	NO	2	NO	NO	NO	ELECTIVE LSCS	3	4.2	3	NO	NO	1
235	PRITHEE	30	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.1	3	NO	NO	1
236	MULLAI	40	PRIM I	3	INSU LIN	NO	3	YE S	YES	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
237	MAITHILI	30	G3P2 L2	4	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.9	3	NO	NO	3
238	SUMATHI	37	G2P1 L1	3	INSU LIN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	3.4	3	NO	NO	1
239	RAJI	28	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1
240	ESTHAR	25	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	4.2	3	NO	NO	3
241	VIJAYALAKS HMI	32	G2P1 L1	4	INSU LIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	3.8	3	NO	NO	1
242	JAYANTHI	26	PRIM I	2	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	1
243	URVASHI	19	G2P1 L1	4	MEA L PLAN	NO	1	YE S	NO	NO	ELECTIVE LSCS	3	3.9	3	NO	NO	1
244	RATHI	30	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	3.8	3	NO	NO	1
245	SANGAVI	24	G2P1 L1	4	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	1
246	THIVANAYA GI	31	G2P1 L1	3	MEA L PLAN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	3.7	3	NO	NO	2
247	MALANI	26	G3P2 L2	5	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	4	3.4	2	NO	YE S	1
248	RAJALAKSH MI	30	G2P1 L1	3	INSU LIN	NO	2	NO	NO	NO	EMERGENC Y LSCS	3	4.3	3	NO	NO	1
249	MANJU PIRYA	20	PRIM I	4	MEA L PLAN	YES	1	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1
250	SHINDU	30	G2P1 L1	4	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.1	3	NO	NO	1
251	PREMA	24	PRIM I	4	MEA L PLAN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
252	SREE DEVI	23	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	3.7	3	NO	NO	1
253	RUBINI	34	G2P1 L1	4	MEA L PLAN	NO	1	NO	NO	YES	LABOUR NATURA	3	3.9	3	NO	NO	1
254	INDRA	25	PRIM I	4	INSU LIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	1
255	JAYANTHI	24	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3	3	NO	NO	1
256	REKHA	39	G3P2 L2	4	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.3	3	NO	NO	2
257	RUBINI	30	G3P2 L2	3	INSU LIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.7	3	NO	NO	1
258	PAVITHRA	24	PRIM I	4	MEA L PLAN	NO	1	NO	NO	NO	INSTRUME NTAL	3	3.8	3	NO	NO	1
259	MARIYA	35	G2P1 L1	4	INSU LIN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	2
260	DEVAKI	30	G2P1 L1	2	MEA L PLAN	NO	1	NO	NO	NO	EMERGENC Y LSCS	3	2.9	3	NO	NO	1
261	KANMANI	34	PRIM I	4	INSU LIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.9	3	YES	NO	1
262	GOMATHI	26	PRIM I	4	INSU LIN	NO	2	NO	NO	NO	EMERGENC Y LSCS	3	4.2	2	NO	YE S	1

263	UJALA	20	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.8	3	NO	NO	1
264	KASIYAMMAL	30	G2P1L1	4	INSULIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	3.7	3	NO	NO	1
265	JERENA	24	G2P1L1	5	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	4	3	NO	NO	1
266	BABY	31	G3P2L2	4	MEAL PLAN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1
267	EGATHA	28	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.3	3	NO	NO	1
268	RAMYA	30	PRIMI	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	1	3	1	NO	YES	1
269	TANUSRI	32	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	1
270	ILAVENI	24	PRIMI	4	INSULIN	NO	3	NO	NO	NO	INSTRUMENTAL	3	4.2	3	NO	NO	3
271	PREETHA	25	PRIMI	4	INSULIN	NO	1	YES	NO	NO	LABOUR NATURA	3	3.2	3	NO	NO	1
272	AISWARYA	37	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.5	3	NO	NO	2
273	SANGEETHA	27	PRIMI	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
274	JIJI	35	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.2	3	NO	NO	1
275	HARITHA	24	PRIMI	5	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	1
276	KALYANI	26	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.2	3	NO	NO	1
277	SHINY	34	G4P3L3	3	INSULIN	YES	2	YES	YES	NO	LABOUR NATURA	3	2.2	3	NO	NO	1
278	SAMYUKTHA	28	G3P2L2	3	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.2	3	NO	NO	1
279	ZARINA	19	PRIMI	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.2	3	NO	NO	1
280	MEENAL	23	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.2	3	NO	NO	1
281	MONISHA	32	PRIMI	3	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	2
282	SUAMTHY	26	PRIMI	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3	3	NO	NO	1
283	LATHA	40	PRIMI	4	INSULIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	3.2	3	NO	NO	1
284	INDIRA	39	G4P3L3	2	INSULIN	NO	1	YES	YES	NO	LABOUR NATURA	3	2.2	3	NO	NO	1
285	RAJESHWARI	24	G2P1L1	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.2	3	NO	NO	1
286	BINU	31	G3P2L2	3	INSULIN	YES	3	YES	YES	NO	LABOUR NATURA	3	2.3	3	NO	NO	1
287	SANKARI	28	G2P1L1	5	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
288	AMALA	25	PRIMI	4	INSULIN	NO	1	NO	NO	NO	INSTRUMENTAL	3	4.2	3	NO	NO	3
289	HARINI	26	PRIMI	4	MEAL PLAN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1
290	VAIDEHI	20	PRIMI	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	4.3	3	NO	NO	1
291	STELLA	30	G2P1L1	4	INSULIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	3.2	3	NO	NO	1
292	MEERA	24	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	3
293	RUDRA	34	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.2	3	NO	NO	1
294	ELIZABETH	19	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.2	3	NO	NO	1
295	RANJANI	30	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.7	3	NO	NO	1
296	KAVYA	28	G4P3L3	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.4	3	NO	NO	1
297	KIRUBA	20	PRIMI	5	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.2	3	NO	NO	2
298	LALITHA	26	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	2.9	3	NO	NO	1
299	AMBIGA	26	PRIMI	3	INSULIN	NO	2	NO	NO	NO	ELECTIVE LSCS	3	4.4	3	NO	NO	1
300	AMMU	32	G2P1L1	4	MEAL PLAN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1
301	KARPAGAM	30	PRIMI	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.2	3	NO	NO	1
302	GAYATHRI	24	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.4	3	NO	NO	1
303	RENUKA	40	G2P1L1	2	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.8	3	NO	NO	1
304	SOUNDARYA	26	G2P1L1	4	INSULIN	YES	1	YES	NO	YES	EMERGENCY LSCS	4	4.3	2	NO	YES	2

305	SHAJINI	19	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.4	3	NO	NO	1
306	SHAHINA	25	PRIMI	4	INSULIN	NO	1	NO	NO	NO	INSTRUMENTAL	3	4.2	2	YES	NO	1
307	KUSHBU	30	G2P1L1	4	INSULIN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.2	3	NO	NO	1
308	ELLAMAL	39	G2P1L1	3	MEAL PLAN	NO	3	YES	YES	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
309	DIILIRANI	31	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.2	3	NO	NO	1
310	VINODHA	23	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.4	3	NO	NO	2
311	KANCHANA	34	G4P3L3	4	INSULIN	NO	1	NO	NO	NO	INSTRUMENTAL	3	4	3	NO	NO	3
312	MUTHAMIZH	26	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	4.2	3	YES	NO	1
313	JASANTHA	28	G2P1L1	4	MEAL PLAN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	3	3	NO	NO	1
314	JANANI	37	PRIMI	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4.2	3	NO	NO	1
315	SHEELA	23	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.4	3	NO	NO	1
316	PRIYAMVADHA	24	G2P1L1	5	INSULIN	NO	3	NO	NO	NO	LABOUR NATURA	3	2.9	3	NO	NO	1
317	KANNAGI	32	G2P1L1	2	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.7	3	NO	NO	1
318	DEVI	26	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	4.4	3	NO	NO	1
319	THENMOZHI	25	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	1
320	MALA	40	G2P1L1	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.7	3	NO	NO	2
321	KARTHIGA	20	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
322	MALAR	26	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	1
323	JANAKI	24	G4P3L3	4	MEAL PLAN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	3.6	3	NO	NO	1
324	VATCHALA	32	PRIMI	4	INSULIN	NO	3	NO	NO	NO	LABOUR NATURA	3	3.8	3	NO	NO	2
325	VASUKI	23	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3	3	NO	NO	1
326	RAMANI	38	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.9	3	NO	NO	1
327	POORNIMA	26	PRIMI	3	MEAL PLAN	YES	3	YES	YES	YES	LABOUR NATURA	3	2.4	3	NO	NO	3
328	ABIRAMI	34	G2P1L1	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.5	3	NO	NO	1
329	SHAKTHI	26	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.8	3	NO	NO	1
330	DATCHAYANI	31	PRIMI	5	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	4.3	2	YES	NO	1
331	BHAVANI	24	PRIMI	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.6	3	NO	NO	1
332	KANTHA	34	G4P3L3	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.7	3	NO	NO	1
333	HEERA	20	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1
334	LINGESHWARI	28	PRIMI	4	INSULIN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	3	3	NO	NO	1
335	VIJYA	26	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	INSTRUMENTAL	3	4.1	3	NO	NO	3
336	DHARANI	23	PRIMI	4	INSULIN	YES	3	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	1
337	VINITHA	24	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.8	3	NO	NO	1
338	MAHALAXMI	38	PRIMI	3	INSULIN	NO	2	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	2
339	ROJA	26	PRIMI	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.9	3	NO	NO	1
340	NANDHINI	28	G2P1L1	4	MEAL PLAN	YES	1	NO	NO	NO	EMERGENCY LSCS	1	2.9	1	NO	YES	1
341	POWN	25	G2P1L1	5	INSULIN	NO	2	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	1
342	PERUNDEVI	19	PRIMI	2	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
343	NAGAMA	24	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3	3	NO	NO	1
344	JYOTHI	28	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.8	3	NO	NO	1
345	RUPALA	34	G2P1L1	4	INSULIN	NO	1	YES	NO	NO	ELECTIVE LSCS	3	3.7	3	NO	NO	2

346	MANONMANI	31	G2P1L1	4	INSULIN	YES	1	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	1
347	DHANALAXMI	26	PRIMI	4	MEAL PLAN	NO	2	NO	NO	NO	EMERGENCY LSCS	3	3.9	3	NO	NO	1
348	NAGAVENI	23	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.7	3	NO	NO	1
349	SHANTHI	20	PRIMI	3	INSULIN	NO	1	NO	YES	NO	LABOUR NATURA	3	2.2	3	NO	NO	1
350	VENNILA	24	PRIMI	4	MEAL PLAN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	3.6	3	NO	NO	1
351	JAYAGOPI	26	G2P1L1	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.5	3	NO	NO	3
352	GAYATHRI	25	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.8	3	NO	NO	1
353	GOMATHI	32	G2P1L1	4	INSULIN	NO	2	NO	NO	NO	ELECTIVE LSCS	3	3.9	3	NO	NO	1
354	VIJAYA	30	G2P1L1	3	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.5	3	NO	NO	2
355	AISHWARYA	28	G2P1L1	4	INSULIN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	2.9	3	NO	NO	1
356	UMA	24	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.6	3	NO	NO	1
357	MAHESWARI	28	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.7	3	NO	NO	1
358	KOTEESWARI	38	G4P3L3	5	INSULIN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	3.8	3	NO	NO	1
359	SUMAYA	26	PRIMI	3	MEAL PLAN	YES	1	YES	YES	NO	LABOUR NATURA	3	2.2	3	NO	NO	3
360	JAYANTHI	23	PRIMI	4	INSULIN	YES	1	NO	NO	NO	EMERGENCY LSCS	3	3.9	3	NO	NO	1
361	MALA	26	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	3.8	3	NO	NO	1
362	VIJAYAM	31	G2P1L1	2	INSULIN	NO	1	YES	YES	NO	LABOUR NATURA	3	2.4	3	NO	NO	1
363	VAITHEGI	24	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.8	3	NO	NO	1
364	VATCHALA	34	G2P1L1	4	INSULIN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	3.8	3	NO	NO	2
365	JANAKI	25	PRIMI	4	MEAL PLAN	NO	1	YES	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1
366	SUMATHY	32	G4P3L3	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4	3	NO	NO	2
367	PRIYA	24	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	ELECTIVE LSCS	3	4	2	NO	YES	1
368	PADMAVATHY	28	PRIMI	4	INSULIN	NO	1	NO	NO	YES	LABOUR NATURA	3	3.4	3	NO	NO	1
369	JAYA	24	PRIMI	4	MEAL PLAN	NO	2	NO	NO	NO	EMERGENCY LSCS	3	4.1	3	NO	NO	1
370	CHITRA	18	G2P1L1	4	INSULIN	YES	1	NO	NO	NO	LABOUR NATURA	3	3.5	3	NO	NO	1
371	MANIMEGALAI	24	G2P1L1	4	INSULIN	NO	3	NO	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	2
372	MAHALAKSHMI	34	G4P3L3	3	INSULIN	NO	2	YES	NO	NO	ELECTIVE LSCS	3	3.5	3	NO	NO	1
373	LINGESHWARI	30	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1
374	LAVANYA	23	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	1	2.9	1	NO	YES	1
375	MALATHY	26	PRIMI	4	INSULIN	NO	3	NO	NO	NO	EMERGENCY LSCS	3	3.6	3	NO	NO	2
376	BAGIYALAKSHMI	20	G2P1L1	5	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	1
377	GEETHA	24	PRIMI	2	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.8	3	NO	NO	1
378	KAMALA	25	PRIMI	4	MEAL PLAN	YES	1	NO	NO	NO	INSTRUMENTAL	3	3.8	3	NO	NO	1
379	JANAKI	26	G2P1L1	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.9	3	NO	NO	1
380	SEETHA	34	G4P3L3	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	4	3	NO	NO	1
381	GOVINDHAMMAL	28	PRIMI	4	MEAL PLAN	NO	2	NO	NO	NO	EMERGENCY LSCS	3	4.2	3	NO	NO	1
382	KOKILA	24	G2P1L1	4	INSULIN	NO	3	NO	NO	YES	EMERGENCY LSCS	3	3.8	2	NO	YES	2
383	LAKSHMI	38	PRIMI	3	MEAL PLAN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.9	3	NO	NO	1
384	DEVI	26	G2P1L1	4	INSULIN	NO	2	NO	NO	NO	LABOUR NATURA	3	3.9	3	NO	NO	1
385	SUCHITRA	32	G4P3L3	4	MEAL PLAN	NO	1	YES	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	1
386	SUJATHA	26	PRIMI	5	INSULIN	NO	1	NO	NO	NO	INSTRUMENTAL	3	3.6	3	NO	NO	1
387	KUMARI	20	G2P1L1	4	INSULIN	NO	1	NO	NO	YES	LABOUR NATURA	3	3.9	3	NO	NO	1

388	DEEPIKA	24	PRIMI	3	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	4	3	NO	NO	1
389	LOGAMBAL	23	G2P1L1	4	MEAL PLAN	NO	1	NO	NO	YES	LABOUR NATURA	4	3.6	2	NO	YES	1
390	SUPRIYA	32	PRIMI	4	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.5	3	NO	NO	3
391	SRIDEVI	24	G2P1L1	5	INSULIN	NO	2	NO	NO	NO	EMERGENCY LSCS	3	3.7	3	NO	NO	1
392	VIMALA	38	G4P3L3	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.7	3	NO	NO	1
393	LURTHAMMAL	24	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	4.4	3	NO	NO	1
394	NANDHINI	25	PRIMI	3	INSULIN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.9	3	NO	NO	1
395	NAVEENA	18	PRIMI	4	MEAL PLAN	NO	1	NO	NO	NO	EMERGENCY LSCS	3	3.8	3	NO	NO	1
396	RUKMANI	34	G4P3L3	4	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	2.9	2	NO	NO	1
397	BAKIYAM	28	PRIMI	4	MEAL PLAN	NO	1	NO	NO	YES	INSTRUMENTAL	3	3.8	3	NO	NO	1
398	KRITHEGA	24	PRIMI	4	MEAL PLAN	NO	2	NO	NO	NO	EMERGENCY LSCS	3	3.6	3	NO	NO	1
399	SUBULAKSHMI	38	PRIMI	5	INSULIN	NO	1	YES	NO	NO	ELECTIVE LSCS	3	3.6	3	NO	NO	1
400	SUGANYA.	32	G4P3L3	3	INSULIN	NO	1	NO	NO	NO	LABOUR NATURA	3	3.6	3	NO	NO	1